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A TREATISE

ON

APOPLEXY,

CEREBRAL HEMORRHAGE,
CEREBRAL EMBOLISM, CEREBRAL GOUT,
CEREBRAL RHEUMATISM,
AND
EPIDEMIC CEREBRO-SPINAL MENINGITIS.

BY

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TO

JOHN J. CRANE, M.D.,

VICE-PRESIDENT OF THE BELLEVUE HOSPITAL MEDICAL BOARD,

This Book is Inscribed

IN TOKEN OF ESTEEM FOR HIS

PRIVATE AND PROFESSIONAL WORTH,

BY HIS OLD FRIEND,

THE AUTHOR.

ON APOPLEXY.

"*Life is short, and the Art long; the occasion fleeting; experience fallacious, and judgment difficult. The physician must not only be prepared to do what is right himself, but also to make the patient, the attendants, and externals co-operate.*"—HIPPOCRATES' FIRST APHORISM.

How eminently fit these pithy sayings of the Father of Medicine are to be the text for a discourse on Apoplexy, is either obvious at first sight, or will become apparent in the sequel.

PREFACE.

M. LITTRÉ says: "*Le mot apoplexie, souvent aussi vague dans la bouche du médecin, que dans celle du malade, se prête à toutes les interpretations de l'ignorance.*" (Vide *Dict. de Méd.;* article, *Apoplexie.*) The late DR. JONES QUAIN remarked that when we look into works on medicine, we are struck with the confusion which exists in the nomenclature of cerebral diseases. (*Mushet.*) Besides, it is generally admitted that we know less concerning the structure, functions, diseases, and morbid anatomy of the brain than of almost any other part of the body.

The following pages have been written because the author believed that he had something of importance to say on the subject of apoplexy. At first, he intended to write only a brief memoir on this topic. Accordingly, some four or five years ago, he began to prepare, from original cases and observations, an article for publication in the *American Journal of the Medical Sciences;* but it soon became so bulky as to exceed even the capacious limits of that large and influential quarterly. The author was therefore compelled either to suppress what he had written, or to publish it in book-form. But, after mature reflection as to which of these courses he ought to pursue, it appeared to him that these cases and observations were much too numerous and valuable, and that they had cost the expenditure of too much time and labor in their collection, to be lightly thrown away. Carefully recorded histories of the ante-mortem phenomena and post-mortem lesions of such cases are still far too few in number; and, indeed, it is probable that we can never make them too numerous, if we expect ever to clear up the obscure points in cerebral pathology and therapeutics. Moreover, this writing constitutes the first instalment of what the author proposes to contribute as the quota of work required from him for illustrating the pathology and treatment of cephalic diseases and injuries. After the above remarks, it is, perhaps, scarcely necessary to say that most of the clinical histories and post-mortem records upon which this book is founded have been drawn up by the author from cases that were seen either by himself or by professional friends, upon whose discrimination and judgment he could rely. Out of *sixty-two* cases which are related, with more or less fulness, in the following pages, *forty-four*, or considerably more than two thirds, are original; and many other original cases are briefly cited.

The change of this work from the form of a magazine article, above mentioned, to that of a treatise on apoplexy and kindred disorders, seemed to require that a full account should be given, not only of the apoplectiform cases, but of the other and more common varieties also, of cerebral hemorrhage, cerebral embolism, cerebral gout, cerebral rheumatism, and epidemic cerebro-spinal meningitis; and this has accordingly been done with considerable minuteness of detail.

The subject of *Infantile Apoplexy* appeared to demand a more thorough exposition than has ever been accorded to it. An entire chapter has therefore been devoted to its elucidation.

The author has also attentively studied the writings of his predecessors and contemporaries on the various topics which are discussed in this volume; and he has freely used the products of their labors whenever it seemed advisable for him so to do. But, at the same time, he has endeavored to give credit in each instance of quotation to whomsoever the credit appeared to be due. He will only add that the preparation of this book has proved interesting and instructive to himself, and that he hopes the book itself will prove interesting and useful to those who read it.

NEW YORK, NOVEMBER 1, 1872.

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Definition.—*Varieties* of Cerebral Hemorrhage.—The phenomena vary considerably in different cases, and the varieties of this affection are several in number.—1st Variety: In it there are no apoplectic symptoms, no loss of consciousness, no coma, nor carus; but hemiplegia is generally present; considerably more than one-third of all the cases belong to it.—2d Variety: In it loss of consciousness or coma occurs, but the symptoms come on gradually, that is, they are not developed with sufficient suddenness to constitute an apoplectic fit; the patient recovers, but remains paralyzed for the rest of his life; this variety also is often met with.—3d Variety: In it the attack soon produces coma which passes away in a short time as in the last variety; but after a few hours consciousness again begins to disappear, and finally is entirely lost; it does not return, and the patient dies comatose.—4th Variety: In it the attack begins with the symptoms of hemiplegia, but the patient is perfectly conscious; later, sopor, coma, and carus occur, and death from paralysis of the medulla oblongata in the end ensues; it often occurs.—5th Variety: In it the hemorrhage occurs during the progress of a fit of epilepsy or eclampsia; two or more cases of this sort will be related in this chapter.—6th Variety: It is the so-called *apoplexie foudroyante* or thundering apoplexy of authors, and is the only form of cerebral hemorrhage which is attended with the symptoms of "stroke" in the proper sense of the word; it is apoplectiform cerebral hemorrhage or hemorrhagic apoplexy, and is comparatively seldom met with.—The last two varieties will claim much of our attention.—*Case* XXIV. 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Hemiplegia of right side with temporary loss of consciousness suddenly occurred without apparent cause; death forty-eight days afterward with symptoms of cerebral softening; autopsy; middle cerebral artery on each side inflamed and occluded by a thrombus; left corpus striatum softened and discolored, etc.—*Cases* XL. and XLI. In both death suddenly occurred with symptoms of intense apoplexy; thrombosis of the basilar artery resulting from inflammation of the walls of that artery was found in both; the inflammatory changes in the arterial coats minutely described; the phenomena of apoplexy, properly so called, may be produced by thrombosis of the cerebral arteries. Finally, the plugging of a cerebral artery in consequence of embolism, or thrombosis, or both combined, occasionally gives rise to cerebral or intracranial aneurism; an instance related.—*Symptoms and Course of Cerebral Embolism;* suspension of the cerebral functions in consequence of arrest in the blood-supply to a portion of the brain; hemiplegia, loss of consciousness, and apoplectic coma; examination of heart and lungs in doubtful cases; symptoms of recovery.—*Case* XLII. Chronic valvular disease of heart; hemiplegia of left side and apoplectic coma suddenly occurred; partial recovery; death about three months afterward from cardiac and renal dropsy; no autopsy; had previously suffered an admonitory embolism of the left brachial artery.—Symptoms of embolism on proximal side of circle of Willis; do. on distal side.—Recovery possible only when the collateral circulation is established in less than forty-eight hours.—Symptoms of pigmentary embolism of the brain.—*Diagnosis of Cerebral Embolism.* 1. The symptoms of hemiplegia and apoplectic stupor when due to cerebral embolism are almost always suddenly developed at the commencement of the attack. 2. Cerebral embolism is always preceded by characteristic premonitory symptoms; they are enumerated. 3. The age of the patient may be an item of some importance.—Pigmentary embolism of the cerebral capillaries occurs only in cases of long-continued malarial intoxication.—*Treatment of Cerebral Embolism;* we cannot remove the obstructing coagula with therapeutic agents; the indications are to favor the development of a collateral circulation without exposing the patient to fresh dangers; we should be very careful about employing venesection in these cases; tonics and nourishing food required much oftener than sedatives and a spare diet; preventive measures that may be employed in cases predisposed to the occurrence of cerebral embolism.

CHAPTER VII.

1. ON NERVOUS APOPLEXY: 2. ON CEREBRAL GOUT, ESPECIALLY APOPLECTIFORM CEREBRAL GOUT, OR GOUTY APOPLEXY: 3. ON CEREBRAL RHEUMATISM, ESPECIALLY APOPLECTIFORM CEREBRAL RHEUMATISM, OR RHEUMATIC APOPLEXY.

1. *Definition of Nervous Apoplexy.*—The possibility of its occurrence shown.—Concerning reflex hemiplegia.—*Case* XLIII. Hemiplegia of left side induced probably by intestinal irritation through reflex nervous action; complete recovery in four days on using aperients, etc.—How reflex nervous irritation may suspend the cerebral functions. The phenomena of surgical "shock" explicable on this hypothesis.—*Case* XLIV.

Graze-wound of left side inflicted by a cannon-ball; immediate death; the only cause of death discerned was "shock."—"Shock" is not synonymous with nervous apoplexy, but its pathogeny is analogous to that of nervous apoplexy.—Reflex nervous apoplexy.—Was Case XVII. an instance of it? Nervous apoplexy from emotional causes; its symptoms described.—Dr. Copland relates a case of nervous apoplexy which occurred in his own practice.—Names of many other observers given who have reported similar cases.—Proximate cause of nervous apoplexy.—The phenomena of epilepsy, except the convulsions, resemble those of nervous apoplexy. 2. *On Cerebral Gout, and especially Apoplectiform Cerebral Gout or Gouty Apoplexy.*—According to Heberden, the gout disposes its victims to apoplexy.—Drs. Garrod, Tanner, Aitken, etc., state that cerebral gout may take the form of apoplexy.—Dr. Copland relates a case of apoplectic gout which occurred in his own practice.—Niemeyer on cerebral gout and gouty apoplexy.—Trousseau relates a case of gouty apoplexy.—He relates another interesting case of cerebral gout.—Gouty epilepsy or epileptiform cerebral gout; a case mentioned by Van Swieten; another case related by Dr. Rounel.—Periodical headache or megrim not unfrequently a form of cerebral gout.—Vertigo sometimes caused by gout; a case mentioned.—Occasionally the special senses, for example, that of sight, are impaired in cerebral gout.—Lowness of spirits, vapors, melancholy, hypochondriasis, and other delusions of a similar character, often due to larvaceous or concealed gout.—Certain disturbances of the cerebral functions usually precede attacks of gout; they are described.—Gout probably occasions insanity, epilepsy, apoplexy, and some other forms of cerebral disease much oftener than is generally supposed.—*Nature of Cerebral Gout;* it is generally a neurosis and not an inflammation of the brain or its membranes.—Cerebral hyperæmia generally present in gouty apoplexy; it is liable to become complicated with cerebral œdema; some gouty swellings of the skin described.—*Treatment of Cerebral Gout;* lowering remedies, and especially the abstraction of blood, to be avoided; when retrocession from some joint has occurred, apply rubefacients to the joint; in bad cases the judicious administration of stimulants often beneficial; general rules for the treatment of anomalous internal gout cannot be laid down; the symptomatic indications in each case should be followed; application of cold to the head, and indications for its employment; potass. bromid., ext. cannabis indica, guaiacum, colchicum, quinia, cerii oxalate, hyoscyamus, opium or morphia, and lithia carb.; the sulphur water of Richfield recommended for cerebral gout.—3. *On Rheumatic Apoplexy and the other forms of Cerebral Rheumatism.*—The subject of cerebral rheumatism has recently attracted much attention.—*Case* XLV. Acute multi-articular rheumatism; sudden death with apoplectiform phenomena; no autopsy, but the head symptoms were not due to embolism nor to intra-cranial inflammation.—Trousseau's case of cerebral rheumatism with sudden death.—Cases reported by Drs. Weber, Murchison, Burdon-Sanderson, and Wilson Fox, in which the temperature rose to a great height before death.—The foregoing cases were apoplectiform in character; the autopsies showed that they were not due to cerebral nor pulmonary embolism, nor cardiac thrombosis, nor to inflammation of the brain or its membranes, but in all probability to the action of the rheumatic poison itself upon the brain.—The predisposing causes of cerebral rheumatism stated.—2. *On the Paralytic Variety of Cerebral Rheumatism.*—Several instances of it mentioned by Trousseau.—3. *On Mental Alienation or Insanity as a Variety of Cerebral Rheumatism.*—Griesinger's remarks concerning it.—Sander has reported five cases of mental disease resulting from rheumatism. Trousseau has related two cases in which chronic delirium was due to cerebral rheumatism.—4. *On the Spasms or Convulsions which are sometimes produced by Cerebral Rheumatism.*—These convulsions may be epileptiform or eclampsic, and tetanic in character.—Cases reported by Drs. Bright, Todd, and Fuller.—5. *On the Choreic Variety of Cerebral Rheumatism.*—What Dr. Bright states, Dr. Sée asserts, and Dr. Trousseau declares concerning it.—A case from Trousseau and another from Thore related.—What Henoch found and H. Roger says concerning the relation between chorea, rheumatism, and cardiac disease.—Dr. Ferber's remarks.—Rheumatic chorea not attended by any structural lesion of the brain or spinal cord.—6. *On the Meningitic Variety of Cerebral*

Rheumatism.—It consists of rheumatic inflammation of the cerebral meninges, and is of rare occurrence.—The occurrence of head-symptoms in the course of rheumatic fever must not always make us think that meningitis has occurred.—A number of cases in point referred to.—How rheumatic meningitis may be recognized during life.—All the varieties of cerebral rheumatism are neuroses except the last.—The occurrence of cerebral rheumatism generally not due to metastasis; and proofs given.—*Treatment of Cerebral Rheumatism:* use of derivatives; the symptomatic indications must be followed; in some cases it should be cooling and sedative, in others analeptic and stimulant; wrapping the body in wet sheets; sulphate of quinine and veratrum viride as antipyretics; venesection should not be employed; utility of opium or morphia, potass. bromid., ammon. bromid., potass. iodid., diffusible stimulants and lemon-juice; conium, Calabar bean, arsenic, and anæsthetics for rheumatic chorea; treatment of rheumatic apoplexy.

CHAPTER VIII.

ON INFANTILE APOPLEXY, AND INFANTILE CEREBRAL HEMORRHAGE.

They differ widely from the corresponding disorders in adults, and occur much more frequently than is generally supposed.—Infantile apoplexy and infantile cerebral hemorrhage are not synonymous terms.—Two statistical tables given to illustrate the frequency with which infantile apoplexy occurs during each year up to five.—Another table to illustrate the frequency with which apoplexy occurs during each period of five years throughout life.—Physiological anatomy of the infantile brain.—Some reasons given why apoplexy occurs so frequently in young children.—The efficient cause or pathogeny of infantile apoplexy described.—*Of Simple Apoplexy occurring in Infancy and Childhood.*—1. It may result from active hyperæmia of the brain.—2. It may result from passive hyperæmia of the brain.—3. It is often met with in pale-looking, badly-nourished children.—*Case* XLVI. Apoplectic stupor suddenly developed in a weakly and ill-fed child of two years; death; autopsy; well-marked anæmia of the cerebral substance; œdema of the membranes, etc., etc.—*Case* XLVII. Chronic marasmus occurring in an infant of four months; purpura hemorrhagica; sudden coma and death; autopsy; anæmia of the brain-substance, and œdema of the membranes; blood thin and watery; mesenteric glands enlarged, etc.—4. Apoplectiform phenomena are sometimes produced in children by latent hydrocephalus.—*Case* XLVIII. Apoplectiform symptoms occurring in a boy eight years old; death; autopsy; found cerebral congestion, copious effusion of serum in the ventricles, etc.—5. Infantile pneumonia may, at the outset, be attended with loss of consciousness and other symptoms of apoplexy.—*Case* XLIX. Apoplectiform phenomena accompanying an attack of pneumonia in an infant of eleven months; death from the latter disease; autopsy; found œdema of the meninges and extensive hepatization of the lungs, etc.—6. Apoplectic coma occasionally supervenes in the course of the inflammatory diarrhœa of infants; an illustrative case related.—Again, infantile diarrhœa sometimes induces thrombosis of the sinuses of the dura mater.—*Case* L. Profuse diarrhœa occurring in a child three months old; stupor, unconsciousness, opisthotonos, and other head-symptoms; death; autopsy; found thrombosis of superior longitudinal and both lateral sinuses; hyperæmia of the meninges, pneumonia, etc.—*Of Cerebral Hemorrhage occurring in Infancy and Childhood.*—Cruveilhier has specially studied the so-called "apoplexy of the newly-born;" his conclusions stated; a brief summary of his cases given; Dr. J. Lewis Smith's observations support Cruveilhier's views; Dr. Smith's cases mentioned; Dr. West's remarks on this subject.—But cerebral hemorrhage may occur at any other period of infancy and childhood.—*Case* LI. Bronchitis (acute) followed by head-symptoms; purpura hemorrhagica; death; autopsy; extensive meningeal hemorrhage, etc.; blood thin, watery, and deficient in plasticity.—*Case* LII. Cachexia, convulsions with great depression, purpura hemorrhagica, and speedy death; autopsy; extensive meningeal hemorrhage; anæmia of the brain-substance, etc.—Another case of passive hemorrhage into the arachnoid related.—Three other cases of meningeal hemorrhage occurring in infants briefly mentioned.—Occasionally the cerebral substance is the seat of extravasation.—Cases reported by Billard, Serres, Wythes, and Richard Quain, referred to.—*Case* LIII. Vomiting, stupor, convulsions, and partial hemiplegia occurring in a girl of eleven years; death; autopsy; found extensive hemorrhage in the cerebral substance and beneath the arachnoid, etc.—*Case* LIV. Scarlatina, anasarca, chronic peritonitis, and pleurisy; death apparently from syncope; autopsy; found thrombosis of the sinuses of the dura mater; four clots of extravasated blood in the brain-substance, etc.—*Case* LV. Diarrhœa and vomiting in a child three weeks old, succeeded by collapse, head-symptoms, cyanosis of the face, and death in three days; autopsy; found thrombosis of the sinuses of the dura mater, extensive hemorrhage into the cerebrum and meninges; anæmia also of pia mater and brain, etc.—*Case* LVI. Typhoid fever occurring in a girl of twelve years; convulsions, purpura hemorrhagica, and death; autopsy; found thrombosis of superior longitudinal sinus; very extensive meningeal hemorrhage; blood extravasated also into the substance of right cerebral hemisphere.—Three other cases in which cerebral hemorrhage was caused by thrombosis of the cerebral sinuses briefly sketched.—*Etiology of Infantile Cerebral Hemorrhage.*—It occurs much oftener in weakly children than in robust ones.—Influence exerted by thrombosis of the cerebral sinuses, and by purpura hemorrhagica in producing it.—Concerning the so-called primary or idiopathic form of cerebral hemorrhage; it occurs but very rarely in young children; Henoch, however, has related three cases.—*Symptoms of Infantile Cerebral Hemorrhage.*—They are very obscure.—Paralysis or hemiplegia but seldom present in such cases.—The reasons given.—Infantile hemiplegia generally due, not to cerebral hemorrhage, but to cerebral tuberculosis.—The symptoms stated which may lead us to suspect the occurrence of cerebral hemorrhage in children.—*Treatment of Infantile Cerebral Hemorrhage.*—When produced by thrombosis of the cerebral sinuses or by purpura hemorrhagica no treatment avails.—How determination of blood to the head or active hyperæmia should be treated; venesection, leeches, purgatives, cold affusion of head.—How the tendency to death may be obviated.

CHAPTER IX.

ON PULMONARY APOPLEXY, (SO CALLED,) OR PULMONARY EXTRAVASATION, AND HEMORRHAGIC INFARCTION OF THE LUNGS, ETC.

Two varieties of pulmonary apoplexy recognized.—But neither of them is attended with the phenomena which characterize apoplexy in the correct sense of the term, except in very rare instances.—To call this affection apoplexy is unphilosophical and absurd.—Remarks of Drs. Trousseau and Gendrin on this point.—The term pulmonary apoplexy has been dropped by the Royal College of Physicians at London, and the term *Pulmonary Extravasation* adopted in its stead.—The new term is quite unobjectionable.—There are two varieties of pulmonary extravasation: 1. The capillary, or circumscribed. 2. The lacerated, or diffused. The capillary variety may be produced, *a*, by diseases of the blood, for example, purpura hemorrhagica, and, *b*, by the lodgment of embolia in the minute branches of the pulmonary artery.—The last species is of frequent occurrence, great practical importance, and has been described under the name of hemorrhagic infarction of the lungs.—*Etiology of Pulmonary Hemorrhagic Infarction.*—It consists in a capillary hemorrhage, confined to a small and sharply-defined section of the lung, and often bounded by the limits of a single lobule.—It is produced by the plugging of small branches of the pulmonary artery with embolia, which are usually brought from some remote part of the body.—The credit of this valuable discovery is due to Virchow.—Embolia often originate in disintegrating thromboses of peripheral veins.—The embolia, which so often produce hemorrhagic infarction of the lungs in cases of heart-disease, come from the right chambers of the heart itself.—The process by which capillary hemorrhage is produced in these cases of pulmonary embolism.—Ludwig's explanation of it.—*Anatomical Appearances produced by Pulmonary Hemorrhagic Infarction.*—Those which result from disease of the heart vary in size from that of a hazel-nut to that of a hen's egg, are blackish-red or almost black in color, completely inelastic, and void of air, so that they feel from without like hard knots.—Old infarctions look paler and yellowish.—Those which are produced by embolia derived from the peripheral veins have generally a small size, (varying from that of a pea to a cherry,) a cuneiform shape, and a superficial situation.—Metastatic pneumonia or abscesses, the result of infarctions, described.—*Symptoms and Course of Pulmonary Hemorrhagic Infarction.*—The symptoms mentioned in detail.—Symptoms of septicæmia or pyæmia, how produced in cases of hemorrhagic infarction of the lungs.—*Treatment of Pulmonary Hemorrhagic Infarction.*—It must be principally directed to the relief of prominent symptoms.—When abstraction of blood should be employed; stimulants administered, and epispastics applied, etc.—*On the Second Variety of Pulmonary Extravasation*, namely, that in which the blood is effused from vessels of some considerable size, the pulmonary tissue is more or less torn or broken down by the extravasation itself, and cavities filled with blood of corresponding size are formed in the pulmonary parenchyma.—*Etiology.*—*Anatomical Appearances.*—*Symptoms.*—The disorder is absolutely deadly and not susceptible of treatment.—The so-called apoplexy of the liver, spleen, kidneys, etc., consists in the extravasation of blood into the substance of these organs.—Such a use of the term apoplexy is also inappropriate and should be abandoned.—They can be designated as renal, splenic, or hepatic extravasations, according to the organ affected by the hemorrhage, with perfect propriety.

CHAPTER X.

ON THE SYMPTOMS, DIAGNOSIS, PROGNOSIS, AND TREATMENT OF APOPLEXY.

1. *Symptoms of Apoplexy.*—The premonitory symptoms described; some that are considered peculiarly ominous; but apoplexy often occurs without warning.—The signs of general plethora stated.—Cerebral hyperæmia often attended with depression of the cerebral functions; symptoms of depression affecting the sensibility; motor symptoms of depression, and physical symptoms of depression enumerated; state of the pupils; state of the pulse and respiration; vomiting.—Cerebral hyperæmia not unfrequently attended with disturbances of the sensibility of an irritating character; they are stated.—In a third form of cerebral hyperæmia the mental symptoms predominate; they are described.—The symptoms which attend the fits of apoplexy are next given in full.—2. *Diagnosis of Apoplexy.*—When the attack occurs in the presence of witnesses, the diagnosis is generally not difficult.—The following pathological states may, at first sight, be mistaken for apoplexy: 1. A state of coma when produced by alcoholic intoxication. 2. A comatose condition when produced by poisoning with opium or morphia. 3. Coma occurring in consequence of injury of the head, such as concussion of the brain, depressed fracture of the skull. 4. Coma when induced by epilepsy or eclampsia, the convulsive movements having ceased before the patient is found, but the symptoms of coma being unabated. 5. Coma the result of uræmia occurring as a consequence of renal disease. 6. Syncope with complete loss of consciousness, the result of fatty degeneration of the heart. 7. Syncope when produced by the direct action of the sun's rays, and therefore occurring as one of the leading varieties of sun-stroke.—How to distinguish apoplexy from each of these affections is pointed out.—Concerning throbbing of the carotids as a symptom of apoplexy; its proximate cause and what it indicates pointed out.—3. *Prognosis of Apoplexy.*—The prognosis of this disease as stated by Hippocrates and Celsus.—Some bad prognostics mentioned.—But the recovery sometimes is rapid and complete in very bad cases of congestive apoplexy.—A case in point related.—4.

Treatment of Apoplexy.—1. On the remedial measures to be employed before the attack, that is, the prophylactic or preventive treatment of apoplexy. It is at this time that the physician can do most good to those who are liable to have apoplexy.—In no other disease are the casual indications of more importance.—The preventive treatment of apoplexy is minutely described.—2. *On the treatment to be employed during the fit of apoplexy.*—After the stroke has occurred the physician is comparatively powerless to benefit his patient.—Then he is practically limited to combating the more dangerous symptoms, or, in other words, to obviating the tendencies to death.—Concerning the employment of venesection and purgatives.—Venesection but seldom advisable, and the indications for its use pointed out.—Concerning the employment of topical bleeding.—The administration of purgatives generally required.—Emetics have been highly recommended; the indications for their employment mentioned.—Concerning the use of repellents and revulsives; cold affusion of the head; the application of the ice-bag or of frozen compresses to the head.—Celsus's remarks.—Dr. West's observations.—The author's experience on this point.—Concerning the use of revulsives or derivatives.—The administration of aconite.—The treatment of *nervous apoplexy* laid down.—The treatment of *infantile* apoplexy discussed.—3. *On the treatment to be employed after the attack of apoplexy.*—The man who has had one fit of apoplexy is strongly predisposed to have another, which will use him still more severely.—What he should avoid is stated.—Concerning the employment of counter-irritants and sedatives in such cases.—They are sometimes very useful.—Blistering was recommended by Sydenham.

CHAPTER XI.

ON EPIDEMIC CEREBRO-SPINAL MENINGITIS, OR SO-CALLED CEREBRO-SPINAL FEVER, SPOTTED FEVER, ETC.

Synonyms.—Definition.—The first outbreak on record referred to.—Many others mentioned.—*Nature of Epidemic Cerebro-Spinal Meningitis;* it is not an idiopathic fever; the proof given.—*Case* LVII. Epidemic cerebro-spinal meningitis; at first several paroxysms of intense pain, located in the spine between the shoulder-blades, occurred; then loss of consciousness, coma, and death suddenly ensued: there were no symptoms of fever whatever; autopsy.—*Case* LVIII. Epidemic cerebro-spinal meningitis; first, paroxysms of extreme restlessness occurred; next, convulsive movements of an irregular character appeared; then delirium, with screaming and much tossing in bed, but without any signs of fever, were present; finally, stupor, with dilatation of pupils, inability to swallow, coma, and death occurred; autopsy.—*Case* LIX. Epidemic cerebro-spinal meningitis; the first symptoms noticed were debility and loss of appetite; next, headache, nausea, thirst, feverishness, and restlessness; then abnormal heat of head, injection of eyes, and delirium; finally, stupor, with dilatation of pupils, coma, carus, and death; no autopsy.—*Case* LX. Epidemic cerebro-spinal meningitis; stupor and lethargy were observed; next, maniacal excitement, screaming, jumping out of bed, and divergent strabismus; then coma, stertor, fits of jactitation, over-action of flexors, and spots of purpura; death by coma; autopsy.—Of these four cases three did not show any symptoms whatever of fever.—When febrile symptoms are present in this disease they are due to the local inflammation and symptomatic of it.—Experience of Niemeyer, Ziemssen, and Githens on this point stated.—Author's conclusions as to the nature of the disease stated.—*Case* LXI. Malignant purpuric fever; febrile symptoms commencing with chill, headache, etc.; on the fourth day numerous hæmic spots appeared; they spread rapidly, and the patient died with symptoms of pulmonary obstruction in about forty hours, the febrile excitement continuing to the last; autopsy; found pulmonary and renal extravasation; skin and mucous membranes extensively blackened with effused blood, etc.—*Etiology of Epidemic Cerebro-Spinal Meningitis.*—1. Influence of age; statistical table.—2. Influence of sex.—3. Occupation and condition in life.—4. It prefers

the hardy, but sometimes attacks the sickly.—*Case* LXII. Epidemic cerebro-spinal meningitis; it followed an attack of diarrhœa; sensations much disordered; death by coma; autopsy.—Two cases observed by Billroth related.—5. Influence of temperature. —6. Is the disease communicable?—7. Is it a form of typhus?—8. Is it a form of paludal fever?—The disease is due to a specific poison; it is a local phlegmasia due to a specific poison.—*Anatomical Appearances or Changes;* they result from inflammation of the cerebro-spinal meninges.—*Symptoms and Course;* these can be fully explained by the changes which the inflammatory process induces in the membranes of the brain and spinal cord; the individual symptoms are considered in detail.—*Diagnosis;* it is not attended with any phenomena which are characteristic; but the diagnosis can generally be made without difficulty; the irregular or exceptional cases are the only ones that give trouble, and they can usually be diagnosticated with certainty by exclusion.—*Prognosis;* the disease is always very dangerous; it is much more fatal in some epidemics than in others, in some parts of the same epidemics than in others, and at some periods of life than at others; each of these points is illustrated; one half of all the deaths have happened before the fifth day; the unfavorable prognostics enumerated; the favorable do.—*Treatment;* the disease is a topical inflammation which has been excited by a specific poison; the therapeutical indications enumerated; the severest cases generally prove fatal, whatever the plan of treatment may be; the remedial measures which have proved beneficial are the local abstraction of blood, the energetic application of cold to the head and spine, the judicious employment of counter-irritants, the liberal use of opium or morphia, combined in some instances with quinine, in others with calomel, but in most cases given by itself; iodide of potassium in the later stages of the disease; inutility of brandy, ammonia, camphor, valerian, musk, sulphite and bisulphite of soda; alimentation of the patient; sanitary measures which should be employed.

APOPLEXY.

CHAPTER I.

SOME GENERAL CONSIDERATIONS RELATING TO APOPLEXY.

Synonyms and Etymology of Apoplexy.—It is a distinct Disease.—*Definition;* that of Aretæus, Celsus, Paulus Ægineta, Sydenham, Boerhaave, and the Author.—Sundry abuses of the term enumerated.—It is not synonymous with cerebral hemorrhage.—Trousseau's views and cases pertaining to this point.—Abercrombie's do.—Mushet's cases of cerebral hemorrhage analyzed.—Conclusions on the point above mentioned.—Apoplexy quite distinct from cerebral hemorrhage.—Objections to the mode in which Cruveilhier uses the term apoplexy.—The remedy.—Dignity of the subject.—Importance of the disease.—Varieties of apoplexy enumerated: 1. Congestive; 2. Serous; 3. Hemorrhagic; 4. Embolic; 5. Nervous.

Synonyms.—GREEK, *ἀποπληξία*; LATIN, *Apoplexia*, *Attonitus Morbus*, *Resolutio Nervorum;* FRENCH, *Apoplexie;* GERMAN, *Apoplexie*, *Hirnschlag;* ITALIAN, *Apoplessia;* SPANISH, *Apoplegia.*

THIS term is derived from the Greek word *ἀποπλήσσω*, which signifies to strike to earth, to knock down; hence the word "stroke" is sometimes used as expressing the same idea. From time immemorial it has been applied to a remarkable group of cerebral symptoms; that is, it has been used to represent a peculiar morbid state or condition of the brain, whereof these symptoms are the exponent; and since they (the symptoms) are of the kind called characteristic, it (the disease) is easily recognized by the clinical observer. Thus, in a clinical point of view, this term stands for a well-defined morbid state of the brain, and in that sense it represents a distinct disease according to the ordinary meaning of the word. Thus, apoplexy has the same right to be considered a separate disease as neuralgia, chorea, epilepsy, tetanus, and several other important affections of the nervous system, whose right to a place in nosography is unquestioned. For what is each of these disorders but a peculiar morbid state of the cerebro-spinal nervous system, or of some part of it, that is characterized, in clinical language, by certain well-defined symptoms or functional disturbances? The expressions clinical observer, clinical point of view, and clinical language are designedly used above because the clinical physician alone is able to point out the difference or make the diagnosis between these nervous diseases, and he can do this only from the symptoms observed during life. The pathological anatomist cannot do it from the lesions found after death. He may, indeed, tell us that in fatal cases of chorea old endocarditis and pericarditis have been met with a certain number of times, or that in fatal cases of epilepsy, sclerosis of the medulla oblongata has been found in a certain proportion of them; but peri-endocarditis is not the essential lesion of chorea, nor sclerosis of the medulla oblongata the essential lesion of epilepsy. So too we shall find in the sequel that cerebral hemorrhage is not the characteristic lesion of apoplexy. Now, while pathological anatomy has not yet shown us what the structural lesions of the brain or other parts of the nervous system are which characterize chorea, epilepsy, tetanus, neuralgia, etc., pathological science in the enlarged sense of the term has at last succeeded in disclosing to us what the morbid state of the cerebral substance is which produces the phenomena of apoplexy, as we shall endeavor to show the reader in our next chapter, which will be devoted to discussing the pathogeny of this disease. We have made the foregoing remarks because it has been suggested in some highly respectable quarters that apoplexy should be banished from nosography, and no longer be considered as a distinct disease, since it may be present in widely different conditions of the brain; and that it should be described in connection with or as a product of these various cerebral lesions. But, laying the further consideration of this topic aside for the present, and taking here the simple ground of expediency only, we may observe that the term apoplexy has been so long in use, is so thoroughly incorporated with the literature, language, and intellectual life of our profession, and, besides, is so extensively employed, so commonly used, and so well understood by the laity, that its exclusion from medical nomenclature is now impossible. Apoplexy must, therefore, on the ground of both justice and expediency, be retained in our nosography, and be described as a distinct disease.

Definition. According to Aretæus[1]: "Apoplexy is a paralysis of the whole body, in respect to feeling, understanding, and voluntary motion." (Vide *Aretæi Cappadocis de Causis et Signis Diuturnorum Morborum*, liber i. cap. vii. p. 84, editionem D. Carolus Gottlob Kühn curavit. Lipsiæ, 1828.) The word apoplexy itself denotes that this paralysis is of sudden occurrence.

According to Celsus[2]: "Attonitos quoque raro videmus, quorum et corpus et mens stupet. Fit interdum ictu fulminis, interdum morbo: ἀποπληξίαν hunc Græci appellant." (Vide *Celsus*, *De Medicina*, liber iii. cap. xxvi.) When freely rendered into English, it reads thus: "We occasionally see patients who have suddenly been stricken down and deprived of their faculties of both body and mind. It is caused sometimes by lightning-stroke, sometimes by disease; the latter the Greeks call apoplexy."

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[1] Aretæus of Cappadocia, a physician who practised at Rome between the reigns of Vespasian and Adrian.

[2] Celsus, Aurelius Cornelius, born towards the end of the reign of Augustus, and still living n the time of Caligula, (from 25–30 B.C. to 45–50 A.D.)

According to Paulus Ægineta[1]: "Apoplectics lie speechless, motionless, and insensible, without fever" (Vide *The Seven Books of Paulus Ægineta*, vol. i. p. 392. Sydenham Soc. translation.) In other words, according to Paulus, the victims of apoplexy are suddenly deprived of consciousness, sensibility, and motility, and lie helpless where they have fallen, without fever.

According to Sydenham[2]: "Profound sleep, utter loss of sense and motion, with the exception of that necessary for respiration. This is labored and stertorous." (Vide *Sydenham's Works*, vol. ii. p. 259.)

According to Boerhaave[3]: "Apoplexia dicitur adesse quandò repentè actio quinque sensuum externorum, tùm internorum, omnesque motus voluntarii abolentur, superstite pulsu plerùmque forti, et respiratione difficili, magna, stertente, unà cum imagine profundi perpetuique somni." (Vide *Boerhaave*, as quoted by Trousseau in his Lectures on Clinical Medicine.) In another work the same writer says: "In apoplexia videtur etiam sui conscientiam deleri, cum omni actione quæ ab anima pendeat, sensu motuque, conservata interim vitali facultate, et homine pro aliquo tempore plantæ quasi vitam vivente." (Vide *Boerhaave*, *Prolectiones Academicæ*, etc., vol. vi. p. 252. Gottingæ, 1744.)

The following is the author's definition: Apoplexy is a disease of the brain which is characterized by the sudden abolition of consciousness, feeling, and voluntary motion, and is produced not by injury, nor by poison, nor by heat, but by some morbid action in the brain itself.

It essentially consists in the sudden occurrence of complete functional inactivity of the cerebrum as a consequence of disease. The patient, if attacked while standing, falls to earth as if stricken down by a powerful blow. He is entirely bereft of consciousness and sensibility, and continues to lie without moving, and utterly helpless, where he has fallen. His breathing is generally labored, deep, slow, and snoring or stertorous. His carotids generally throb with more or less violence. His face is often seen to be turgid with blood, his pupils contracted, and his conjunctivæ injected. But in other cases the face is pale and the pulse feeble from the beginning of the fit. Such are some of the more striking symptoms of apoplexy in the ancient and clinical sense of the term.

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[1] **Paulus Ægineta lived in the seventh century, (A.D. 660.)**

[2] **Sydenham, Thomas, born about the year 1624, and died in 1689.**

[3] **Boerhaave, Hermann, born in 1668 and died in 1738.**

At the present day, however, the word apoplexy is employed by many writers to signify interstitial hemorrhage, and every other form of extravasation of blood which occurs suddenly in the substance of any organ or tissue. Hence they speak of cerebral apoplexy, pulmonary apoplexy, etc. etc. Formerly it was always—and still is by many—used in a restricted sense, to signify the train of phenomena which characterize cerebral apoplexy. (Vide *Dunglison's Medical Dictionary.* Art. Apoplexy.)

Thus, in recent times,[1] this term has come to be used by different persons in quite different senses. When pathological anatomy began to be cultivated, it was found that cerebral hemorrhage was not unfrequently present in cases belonging to the category of apoplexy, and as the views and modes of expression of the pathological anatomists came to preponderate, the term came to be synonymous with not only cerebral hemorrhage but with parenchymatous hemorrhage in every other organ of the body also ; so that, besides apoplexy of the brain, we have apoplexy of the lungs, liver, spleen, kidneys, stomach, intestines, bladder, trachea, larynx, heart, voluntary muscles, marrow of the bones, and, indeed, of every other structure into which blood may chance to be extravasated. Thus, we find this term commonly used to-day in two very different senses ; the one being the ancient clinical, and etymologically correct, and the other the purely pathologico-anatomical. Out of this looseness and inexactness in the use of the word apoplexy has grown confusion, inaccuracy, and serious misapprehensions, things which are always unseemly and detrimental to progress, especially in matters pertaining to science. This evil ought not to be tolerated any longer. It appears to the author that the time has now come when the term apoplexy should either be very much restricted in its use, or else that a new terminology should be invented.[2] It seems to him, however, to be by far the better plan to restrict the use of this term within proper limits ; that is, to employ the word apoplexy only in accordance with its ancient meaning, and to make use of other terms—such, for example, as "extravasation" or "effusion of blood," and, in a certain class of cases, the phrase "hemorrhagic infarction"—to represent the parenchymatous hemorrhages which occur in the various organs and tissues of the body; and he doubts not that others, when they reflect on the subject, will be of his way of thinking. This topic, however, will be suitably discussed further on, in the chapter on so-called pulmonary apoplexy. The kind of hemorrhagic cases to which "hemorrhagic infarction," and the other kind to which the word "extravasation" should be applied will there be pointed out.

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[1] Since the close of the last century.

[2] The sagacious Bennett remarks : "The term apoplexy has been used in two senses. By the older writers and clinical observers, it was used to denote a sudden loss of consciousness and volition, independent of the various morbid lesions which may occasion these symptoms. By the followers of the French school of pathology the same word has been applied to an extravasation of blood into an organ ; and hence the terms apoplexy of the lung, apoplexy of the spinal cord, apoplexy of the kidney, etc. Ecchymosis of the skin, the result of contusion—such as is present in what is commonly called a 'black eye'—is in this sense an apoplexy. I believe it better," he says, "to adhere to the meaning of our ancestors, the more so as it is not always possible to determine when a cerebral hemorrhage is present. Hence the two cases which have been given, characterized by sudden loss of consciousness and volition, coming on spontaneously, are called apoplexy. But should such cases prove fatal, and we are enabled to speak positively as to the cause of the apoplexy from post-mortem examination, then we may denominate them with more propriety cerebral hemorrhage, softening, etc., etc., according to circumstances." (Vide *Bennett's Clinical Lectures on the Principles and Practice of Medicine*, pp. 400, 401, 5th Am. ed

The term apoplexy in its clinical or correct sense, at least so far as practical matters are concerned, is not by any means synonymous with even cerebral hemorrhage. On the contrary, cerebral hemorrhage is attended but seldom, comparatively speaking, by the symptoms or phenomena which characterize a fit of apoplexy, and frequently not one of the symptoms of apoplexy, properly so called, is present in cases of cerebral hemorrhage. Extravasation of blood into the substance of the brain generally occasions paralysis in the form of hemiplegia, on the opposite side of the body, and the extent or degree of this paralysis is determined by the part of the brain involved, and by the quantity and suddenness of the effusion; but this hemiplegia is not necessarily accompanied by the group of symptoms which characterize an apoplectic fit, nor indeed by any of these symptoms. Take, for example, a case of cerebral hemorrhage which, for more than two years, has been under my own observation. This patient is a gentleman of about 55, of strong constitution, full habit, and, before the attack, of a free way of living. He was seized with cerebral hemorrhage, in the evening, while on his way home from business. He was riding in a coach alone at the time. He noticed that he felt somehow strangely, as if he were sliding off from a house-roof, and as he had never felt before; but did not lose consciousness, or, if he did at all, it was only for a moment. He thinks, however, that he did not lose himself at all. His bad feelings soon passed away, and he did not suppose any thing serious was the matter until he endeavored to get out of the coach on reaching home. Then he found himself helpless because he had lost the use of his left arm and leg. He was lifted out of the coach and carried up-stairs to his room. His speech was muttering and indistinct, but his mind was clear. There was strongly-marked hemiplegia on the left side. Besides the arm and leg, his face also was paralyzed on that side. His mouth was drawn considerably upward and outward toward the right ear. His tongue when protruded deviated toward the left side. There was much diminution of sensibility, as well as loss of motility generally, on the affected side. The symptoms and course of the hemiplegia were so characteristic that there is no room for reasonable doubt but that the paralysis was occasioned by an extravasation of blood in the right cerebral hemisphere. On the second and third days he had considerable fever, but it soon subsided under simple treatment. The anæsthesia rapidly diminished and in a few days disappeared. He also recovered pretty rapidly from the motor-paralysis. At the end of a week or ten days, the facial palsy was no longer perceptible, and he had considerable use of the paralyzed leg, although it was still very weak and disposed to bend under him. In the arm, however, the palsy disappeared much more slowly. After the lapse of eighteen months, he had not fully recovered the use of this member, for it still remained deficient in muscular power, although its sensibility was completely restored. It is interesting to note that all the phenomena in this case were such as belong to cerebral hemorrhage, and to it alone. The hemiplegia, the secondary fever, the symptoms of rapid improvement, and the incomplete recovery, exactly correspond to a moderate extravasation of blood in the right cerebral hemisphere, to traumatic inflammation of the brain-substance around the clot, and to absorption of the serum, etc., without, however, the complete removal of the clot. This then, beyond a reasonable doubt, was a case of cerebral hemorrhage. It should also be stated that when the attack occurred not only was the patient not deprived of consciousness, as he believes, but he did not fall off from his seat in the coach; nor was he shaken off from or down in his seat by the severe jolts and bounces incident to driving across railroad tracks and over rough pavements, as he would have been if his sensorium commune were much affected by the attack. Here, then, is a case of cerebral hemorrhage entirely unattended with the phenomena of apoplexy. Nevertheless, according to the terminology in general use, this man had an attack of sanguineous apoplexy. Apoplexy without any of the symptoms of apoplexy; what an absurdity! The testimony of Dr. Trousseau, lately the learned and eloquent physician at the Hôtel-Dieu of Paris, is also very strong on this point. In one of his charming lectures on clinical medicine, he has forcibly exposed this absurdity into which we have fallen in recent times. He says: "During the period that I have been in the habit of delivering clinical lectures at the Hôtel-Dieu of Paris, I have only seen one female and two male patients in whom cerebral hemorrhage *seems* to have set in at once with apoplectiform phenomena." (Vide *Lectures on Clinical Medicine*, vol. i. p. 4. New Sydenham Soc. translation.) He states that one of them was a "rag-collector who was found in the streets, and brought to the hospital in the most profound stupor." . . . "He died on the second day after his admission, and when his brain was placed on the amphitheatre table, I announced to you [that is, his clinical class] that we should find an effusion in the ventricles. It turned out that the blood had been first poured out into one of the corpora striata, from there had passed into the lateral ventricle of the same side, and after filling it, had broken down the septum lucidum, and got into the other lateral ventricle." The second of these three cases was "a woman aged 63, who had had, the preceding year, a so-called paralytic stroke." . . . "This time, she was found in bed in a state of profound coma. She died without having been roused, and, as in the last case, there was found, in addition to the remains of the small hemorrhage of the previous year, an enormous clot, beginning in one optic thalamus, and distending both lateral ventricles." The last of these three cases was a young man, "who, whilst presenting all the symptoms of encephalitis, was suddenly seized with epileptiform convulsions, and died a few minutes afterwards in a state of carus. In this case there was hemorrhage in the pons Varolii, which had made its way into the fourth ventricle, and ruptured the valve of Vieussens." (*Ibid.*, vol. i. pp. 4, 5.) Further on, he says: "I made use, just now, gentlemen, of a very restricted form of expression, when speaking of the rag-collector who had been picked up in the streets in a state of *apoplexy*. I told you that the cerebral hemorrhage *seemed* to have set in with apoplectic symptoms, as if I had some reason for doubting the accuracy of the fact. I doubt it, indeed, for if it be undeniable that the man was picked up and brought to the Hôtel-Dieu in a state of apoplexy, and that the old woman I just mentioned was found one morning in her bed in a state of coma, who can positively affirm that these symptoms of apoplexy set in all of a sudden?" . . . "Who knows how the attack set in? Who knows whether for half an hour, an hour, or even more, the symptoms had not run the same slow and progressive course as in the three cases [of cerebral hemorrhage, other than those now under consideration, which] I have just related to you? Nay, I add that this is infinitely probable, if not absolutely certain. The reason why I speak so positively is, because for more than fifteen years my attention has been directed to this point in the history of cerebral hemorrhage, and I never had the chance, *never once*, of seeing a patient struck down suddenly by *apoplexy*, in the classical and etymological sense of the word. I have not seen a single case in my hospital or my private practice, or in the practice of my professional brethren who have done me the honor of asking me to meet them in consulation. I have, indeed, seen a great number of individuals suffering from cerebral hemorrhage, in the most profound apoplectic stupor; but in every case, *without exception*, when the attack has occurred in the presence of witnesses, it had come on gradually, and had in general been slight at the outset, coma supervening in ten minutes, half an hour, an hour, or several hours afterwards; but in no single instance, I repeat, have I seen a man with cerebral hemorrhage struck down as by a blow, and dropping instantly in a state of unconsciousness." (*Ibid.*, vol. i. pp. 6, 7.)

"Under certain circumstances only," he continues, "is this the case, and I hasten to make the statement, lest my views should be deemed exaggerated or singular. The patient" . . . [the young man] "who died of hemorrhage into the pons Varolii and tearing of the valve of Vieussens, became suddenly comatose, and remained so until his death, which occurred shortly after. But what did his night attendant tell us? The patient, you remember, had acute encephalitis, that would have carried him off a few days later had not this unforeseen attack occurred. All of a sudden he is seized with epileptiform convulsions, and he dies a few minutes afterwards, without having been aroused from the most profound apoplectic stupor. Note well, gentlemen, that to the ordinary phenomena of hemorrhage there was superadded, in this case, an attack of convulsions, which alone, and apart from all complications, suffice for producing apoplectic stupor. I admit, then, that whenever cerebral hemorrhage begins with an epileptiform attack, apoplectic stupor will set in suddenly, as it does after every attack of epilepsy. I will add further, with regard to this case, that the hemorrhage was seated in the pons Varolii, that is, in a point where all the nerve-fibres converge. When hemorrhage occurs in a part so esential to life, I understand the suddenness of apoplectiform phenomena. But again, I repeat, apoplectic stupor is a very exceptional symptom of invasion in cases of cerebral hemorrhage, unless there be lesion of a central part, or an attack of convulsions." (*Ibidem*, vol. i. pp. 7, 8.) "I make no exception," he adds, "even in favor of blood-effusion into the lateral ventricles. Before this happens, the blood has accumulated in a portion of the brain, near the surface of the ventricles, and has already given rise to symptoms which may have been mistaken, but which indicate, to the experienced practitioner, the existence of hemorrhage, or of a morbid process which has caused capillary hemorrhage. Suppose, for example, that such a morbid process takes place in a corpus striatum, and that in consequence of it a number of small clots have formed, varying from the size of a small pin's head to that of a small lentil, so far there will only be a sensation of weight in the head, and of numbness in the side opposite to the lesion; but if, all of a sudden, on the blood finding its way into a ventricle, the person falls down, struck with apoplexy, the symptoms noticed before the occurrence will be considered as premonitory only, whilst they were in reality symptoms of a simple or multiple hemorrhage, dating a few days back. In such a case, the hemorrhage is supposed to occur only when the patient becomes apoplectic; whereas the blood is effused into the cerebral substance at the time the first symptoms manifested themselves, the subsequent formidable accidents being caused by the sudden irruption of the blood into the ventricles. (*Ibid.*, vol. i. pp. 7, 8.)

In another part of the same lecture, Trousseau declares with the authority of a master: "I repeat, gentlemen, apoplexy proper is very rare in cerebral hemorrhage." (*Ibidem*, vol. i. p. 5.) Trousseau's opinions on this subject are entitled to great weight on account of his talents, learning, industry, and vast experience in both hospital and private practice. They could not well have been given here in smaller space with justice to their distinguished author.

The cases of cerebral hemorrhage which Dr. Abercrombie has published strongly corroborate Trousseau's views. The following are presented as examples: 1. A clergyman, aged 55, while preaching was observed to stop and put his hand to his head; he then attempted to go on, but talked indistinctly, and had evidently lost his recollection; he supported himself by grasping the side of the pulpit. Assistance being immediately given, he was found speechless and paralytic of the right side, but appeared to be sensible. He rapidly became more and more oppressed, and, in about twenty minutes from the attack, was entirely comatose. He died in about twenty-four hours. There was an extensive extravasation of blood found in the left hemisphere of the cerebrum, which had burst into the left ventricle, and after distending it had torn through the septum into the right ventricle. (Vide *Abercrombie on Diseases of the Brain*, etc. p. 221, 4th ed. Edinburgh, 1845.)

2. A man, aged 32, of very full habit, was suddenly seized with intense headache, followed by vomiting. In a few minutes, he began to talk incoherently, and soon after fell down insensible with a slight convulsion; face extremely pale, body cold, pulse scarcely to be felt. From this state, which resembled syncope, he passed gradually into the appearance of apoplexy, and three hours afterwards his breathing was stertorous. He now lay in profound coma, and died twenty-nine hours after the attack. All the ventricles of his brain were found distended with clotted blood, which had burst into them from a laceration in the substance of the cerebrum. (*Ibid.*, p. 222.)

3. A lady, aged 40, of spare habit, was suddenly seized with headache, accompanied by vomiting and diarrhœa. At the same time she began to talk incoherently. She continued to talk incoherently for two hours and then sank into profound coma. She died fourteen days afterwards of this affection of the brain. In the substance of the right hemisphere of the cerebrum was a cavity containing a clot of blood the size of a hen's egg, and communicating with the right ventricle. There was another cavity filled with extravasated blood in the left hemisphere. All the

ventricles were full of dark-colored liquid resembling coffee. (*Ibid.*, pp. 227, 228.)

4. A gentleman, aged 55, of thin spare habit. The third and fourth ventricles of his brain were found full of coagulated blood, and in the lateral ventricles was a considerable quantity of bloody serum. He was not at first insensible, although he gradually became so. First he complained of giddiness and sickness; next of impairment of muscular power; next he became partially comatose; finally he became completely comatose, and died in a few hours. (*Ibid.*, pp. 229, 230.)

Abercrombie's views bear a remarkable resemblance to those which have been quoted from Trousseau. He commences the section in which his cases of cerebral hemorrhage that proved fatal are mostly presented by saying: "The cases to be described under this section differ remarkably from those which are properly styled apoplexy. They are not at first apoplectic; or, if there be at the very first attack loss of sense and motion, this state is recovered from in a few minutes, or perhaps seconds, without any remedy." "The patient continues for some time, perhaps an hour or two, cold and feeble." "He is quite sensible, but oppressed." "By degrees he recovers heat and the natural appearance of the countenance, and the pulse improves in strength. The face then becomes flushed; he is more oppressed; he answers questions slowly and heavily; and at last sinks into coma, from which he never recovers. The period occupied by these changes varies exceedingly." "In one case . . . there intervened from the first attack to the commencement of coma, five hours, in a second, twelve hours, in a third, three days, and in a fourth, not more than fifteen or twenty minutes." "Sometimes the coma follows so speedily, that the case closely borders upon the attack of simple apoplexy. But I think [he says] there is almost always a certain interval of sense, generally with violent complaint of pain, and not that immediate and complete loss of sensibility which occurs in what may be strictly called the apoplectic attack." (*Ibid.*, pp. 217, 218.)

The clinical observations of other writers support the views of Abercrombie and Trousseau. The next two cases are taken from a recent work by Mushet. 1. E. S., female, aged 72, attacked about 8 P.M. while in the water-closet. On arriving, Mushet found her in bed. She was conscious but could not answer questions well. She stated that she had never suffered from headache or giddiness, but now had pain over the right brow and temple. There was loss of power on the left side, but she could raise, with some effort, her left leg from the bed, and also, with more difficulty, her left arm. Mouth drawn slightly to the right; respiration easy and natural; pulse slow and compressible; radials calcified; pupils contracted; arcus senilis marked. Insensibility came slowly on, and she died at 1 A.M., stertor and convulsive movements of the paralyzed side having previously ensued.

The right ventricle of the brain was found completely filled with black coagula; right corpus striatum and thalamus extensively lacerated; clotted blood also found in the left, third, and fourth ventricles, and at the base of brain; the coagula were altogether about 2 oz. in weight. (Vide *A Practical Treatise on Apoplexy,* (*Cerebral Hemorrhage,*) *etc., by William Boyd Mushet,* M.B., pp. 28, 29. London, 1866.)

2. J. G., male, aged 63, who already had had two attacks of paralysis, in neither of which, according to his sons, was he insensible, but ever afterwards his speech was indistinct, and the use of his left side impaired, was again attacked about 2 P.M. He was seen to stagger by another man, who saved him from falling. When Mushet was called, he could speak imperfectly and sit without support. At 8 P.M. he was insensible, breathing heavily and slowly, but not stertorously; pupils contracted and insensible to light. At midnight his breathing was becoming stertorous, and his mouth drawn to the right side. He died at 7 A.M.

On section of right hemisphere of cerebrum a large clot was exposed before reaching the level of the ventricles. It had completely destroyed the right thalamus and posterior part of the corpus striatum. It had also broken up and lacerated the surrounding brain-substance so as to extend nearly to the surface of the hemisphere. It was black, chiefly solid, and weighed 2¾ oz. It also extended into the third and fourth ventricles. (*Ibid.*, pp. 23, 24.)

Mushet relates in all ten cases of cerebral hemorrhage. The subjects were all aged or well advanced in years. In every instance the extravasation was copious and proved quickly fatal. In two of these ten cases, the symptoms of coma were gradually developed. (These cases have just been presented to the reader.) In six instances, it is not known whether the symptoms were gradually developed or not; for the subjects were either dead or dying when found, and therefore the commencement of their attacks had been unobserved. In the remaining two cases, however, insensibility suddenly occurred. One fell down in a fit and died in about two hours; the other suddenly dropped dead in the street. So then of Mushet's ten cases of cerebral hemorrhage, only two can be fairly claimed as instances of apoplectic fits, and it is probable that even in one of them the attack began with epileptiform convulsions, and that the insensibility was produced at first, not by the cerebral hemorrhage, but by the epileptiform convulsions. We may here also remark that in but one of the nineteen cases of cerebral hemorrhage related by Abercrombie did the attack set in with the symptoms of apoplexy.

From the clinical and post-mortem observations related above, we draw the following conclusions:

1. That apoplexy and cerebral hemorrhage are not synonymous terms. 2. That in occasional instances, cerebral hemorrhage does begin with apoplectic stupor, and then the disease should be called hemorrhagic apoplexy, or apoplectiform cerebral hemorrhage. The lesions which characterize these cases will hereafter be considered in the chapter especially devoted to hemorrhagic apoplexy. 3. That cerebral hemorrhage often runs its course without producing any apoplectic symptoms whatever, or, in other words, cerebral hemorrhage is not necessarily accompanied by apoplectic symptoms. Generally, these symptoms do not show themselves unless the extravasation is profuse. Small hemorrhagic clots can be formed, not only without the patient presenting any of the phenomena of apoplexy, but without his having any impairment of intellect, or any affection of the senses; in fact, without any symptoms indicating that the brain has been deeply modified in its functions. The only symptoms which then characterize the case are those of paralysis (hemiplegia) more or less complete, and more or less limited in extent on the opposite side of the body. (*Trousseau.*)

Apoplexy in the classical and real sense of the term is generally connected with pathological conditions of the brain which are widely different from cerebral hemorrhage. For instance, it not unfrequently results from sudden and excessive hyperæmia (congestion) of the brain as it does in cases of the so-called ictus sanguinis, several original examples of which will be found related in the chapter on congestive and serous apoplexy. Again, it sometimes results from cerebral anæmia when suddenly produced and extensive in scope, as it does in those cases of cerebral embolism where a cerebral artery of large size becomes suddenly occluded by a clot of blood or plug of fibrine swept into it by the torrent of the circulation from some distant part, and where a large portion of the brain becomes, in this way, suddenly deprived of its supply of nutrient blood; or as it does in those rare cases where mental emotions of a powerfully depressing nature, such as terror, grief, and despair, produce syncope and death—emotions which suddenly whiten or render anæmic not only the face but the substance of the brain also. Finally, apoplexy is not unfrequently occasioned by rapid and profuse effusion of serum, especially when it is associated with œdema of the substance of the brain. A number of original cases belonging to this category will also be found related in the chapter on congeṕtive and serous apolexy. Thus, we perceive not only that cerebral hemorrhage is but seldom attended by the phenomena of an apoplectic fit; but also that when an apoplectic fit does occur, it is almost always occasioned by some lesion of the brain that is quite distinct from cerebral hemorrhage. This statement of facts is obviously of special value to the clinical observer and the practical physician, that is, to those who have to deal with and interpret the symptoms of brain-disease.

Cruveilhier and those who follow him, for example Rochoux, Littré, and Mushet, have restricted the use of the term cerebral apoplexy to the cases in which hemorrhage occurs spontaneously in the brain. (Vide *Anatomie Pathologique du Corps Humain*, tome i. liv. 3, p. 2; *Dictionnaire de Médecine*, art. Apoplexie; and *Mushet's Treatise on Apoplexy*, p. 4.) Now, such a use of this term would not be objectionable if cerebral hemorrhage were generally attended by the phenomena of apoplexy. But such is not the fact, for we have conclusively shown that cerebral hemorrhage very seldom begins with apoplectic stupor. The symptoms, in even the worst cases, are generally developed one after another, as they were in the cases which we have quoted from Abercrombie and Mushet; and Mushet himself admits that only in very rare instances does the patient suddenly stagger and fall down insensible as in apoplexy. "More commonly he becomes soporose and gradually merges into coma." (Vide *Treatise on Apoplexy*, (*Cerebral Hemorrhage*,) etc., pp. 96, 97. London, 1866.) Besides, cerebral hemorrhage very often occurs without producing any apoplectic phenomena whatever, as for example, in the cases of hemiplegia from extravasation that are so frequently met with, wherein the patient has never lost his consciousness. Finally, as we have just shown, the apoplectic stroke or fit, in a vast majority of instances, is due to pathological states of the brain which are quite distinct from extravasation of blood. Under these circumstances, to accept Cruveilhier's definition of apoplexy would be to sanction a monstrous perversion of language. It would involve the taking away of the name apoplexy from the cases which really present the apoplectic phenomena, and the bestowing of it upon a disorder which generally is not attended with apoplectic symptoms. No wonder, then, Littré complains that "The term apoplexy is frequently as vague in the mouth of the physician as in that of his patient, and readily lends itself to all the misconceptions arising from their ignorance." (Vide *Dictionnaire de Médecine*, art. Apoplexie.) But the remedy is easy. Restore the word apoplexy to its original meaning—the only meaning which it had for more than twenty-two centuries—use it in no other sense, and apply to the disorder now often called "cerebral hemorrhage" the term "cerebral hemorrhage" in our nosology, and all vagueness will disappear. Such a course is eminently philosophical. It will also satisfy the needs of both the clinical observer and the pathological anatomist.

Dignity of the subject.—Apoplexy is a disease which occurs too frequently, and its symptoms are altogether too striking in character, not to have been observed even in primitive states of society. It was well known to the ancient Greek and Roman schools of medicine. Hippocrates, Celsus, Aretæus, Galen, Paulus Ægineta, and the Arabian writers of the middle ages, speak of it; and, in all probability, long before the time of the physician of Cos, who was born about 460 years before the Christian era, it was mentioned in writings that have not come down to us, and by authors whose names have been entirely forgotten.

This disorder possesses great importance in a practical point of view. In the first place, it is very fatal. A very large proportion of those who are attacked with it, die. Hippocrates says: "It is impossible to remove a strong attack of apoplexy, and not easy to remove a weak attack." (Vide *Aphorisms*, sec. ii. 42, *Works of Hippocrates, translated by Dr. Adams*, p. 713, Sydenham Soc. ed. London, 1849.) Celsus says: "Isque morbus mediocris vix sanatur, vehemens sanari non potest." (*De Medicina*, lib. ii. cap. viii.) Aretæus in a paragraph which we have already referred to, says: "Apoplexy is a paralysis of the whole body in respect to feeling, understanding, and voluntary motion; wherefore it happens that a strong attack of this disease cannot be removed in any way, and a weak one only with much difficulty." (Vide *Aretæi Cappadocis de Causis et Signis Diuturnorum Morborum*, liber i. cap. vii. p. 84, editionem D. Carolus Gottlob Kühn curavit. Lipsiæ, 1828.) The statements of Hippocrates and Celsus and Aretæus concerning the gravity of apoplexy are as true to-day as when they were originally uttered. In the second place, this disease is often met with. During the year ending December 31, 1869, it destroyed 398 lives in the city of New-York, and 107 lives in the city of Brooklyn, as shown by the official report of the Sanitary Superintendent for that year. In other words, 1.58 per cent of all the deaths that occurred in New-York, and 1.21 per cent of all the deaths that occurred in Brooklyn, during the period above mentioned, were occasioned by apoplexy. Prior to the establishment of the Metropolitan Board of Health, the City Inspector's reports show that, during a period of eight years ending with Dec. 31, 1865, no less than 2487 persons perished of this disease in the city of New-York alone. Thus, we find the average yearly mortality from apoplexy in New-York for this period to have been 310, which, considering the increase of population, corresponds very closely with the results obtained by the more careful and exact mode of registration that was inaugurated by the Metropolitan Commissioners of Health. There are but two diseases of the nervous system which prove more destructive to human life than apoplexy. For example, the very able report by Dr. Elisha Harris for the year 1869, mentioned above, shows that while 398 persons perished of apoplexy in a twelvemonth, 1000 died of convulsions, and 725 of meningitis during the same period. Apoplexy, therefore, stands third in the list. But, inasmuch as convulsions and meningitis are *par excellence* disorders of infancy, and apoplexy belongs mainly to the adult period of life—for the report shows that nearly all the cases of convulsions and meningitis occurred in infants or children less than five years old, and that comparatively but a small portion of the cases of apoplexy occurred in infants or young children—it follows that apoplexy is the principal disease of the nervous system which destroys life during the adult period. No other affection belonging to that class of disorders is anywhere near so destructive to grown people as apoplexy. Furthermore, it is not improbable that some of the cases reported to the Bureau as fatal from congestion of the brain (there were 208 such cases reported in 1869, of which about one third occurred in adults) were in reality cases of so-called congestive apoplexy, since this disease is almost exactly synonymous with that form of extreme cerebral congestion which proves suddenly fatal. Thus it becomes highly probable that the number of deaths from apoplexy in the city of New-York reported by the Sanitary Superintendent for 1869, is not over-estimated, although at first sight the figures may seem to be large. It is hardly necessary to state that cases of cerebral apoplexy only are here referred to.

Varieties of Apoplexy.—In order to promote clearness of statement and likewise to facilitate the description of this disease, we shall consider it under each of the following heads: namely,

1. Congestive apoplexy, so-called, which embraces, as its name implies, the cases wherein hyperæmia of the brain or its membranes is found after death.

2. Serous apoplexy, so-called, which includes the cases wherein an effusion of serum of more or less abundance is found either in the ventricles or in the membranes or in the substance of the brain, or in all of these situations, on examination after death. The serous form of apoplexy, however, is closely allied to the congestive, and in most cases is nothing more than a variety of it. The relationship which exists between serous and congestive apoplexy will be more fully shown when we come to study these affections in detail. These two classes taken together constitute what is known to many as simple apoplexy.

3. Hemorrhagic apoplexy, or apoplectiform cerebral hemorrhage. It includes the cases of spontaneous extravasation of blood into the substance or upon the surface of the brain that are attended by the phenomena of an apoplectic stroke. These cases are not of very frequent occurrence as we have already shown, but their importance will render it necessary for us to discuss the causes, symptoms, course, and consequences

of cerebral hemorrhages in general under this head. This affection is also called sanguineous apoplexy.

4. Embolic apoplexy, or apoplectiform cerebral embolism. It consists of the cases, in which the symptoms of apoplexy are produced by the sudden plugging up of one or more of the cerebral arteries having considerable size, or of a great number of the cerebral capillaries simultaneously with embolia, whereby a large part of the brain is suddenly deprived of its blood-supply and made anæmic.

5. Nervous apoplexy. It embraces the cases in which the symptoms of undoubted apoplexy have been present during life, and after death no material lesion has been found in any part of the body to account for them.

But in addition to these varieties of apoplexy, we shall have occasion to speak of meningeal apoplexy, the apoplexy of new-born infants and children, and the gouty and rheumatic forms of this disease.

It is probable, however, that in all these forms or varieties of apoplexy, as they are called, the efficient cause of the disease is in reality the same. Its proximate causes in the shape of organic lesions of the brain or its blood-vessels, or its membranes, may vary much in different cases without requiring any real difference so far as the efficient cause is concerned, as we shall show in the sequel.

CHAPTER II.

ON THE PATHOGENY OF APOPLEXY.

Congestive and hemorrhagic apoplexy generally supposed to be due to abnormal pressure of the blood on the brain.—Something more than cerebral hyperæmia, however, is necessary to produce congestive apoplexy.—Cerebral anæmia, for example, from embolism of the cerebral arteries, often induces the symptoms of apoplexy.—Niemeyer has shown that intra-vascular pressure is not the cause of cerebral paralysis or apoplexy.—In congestive apoplexy the phenomena are due to want of fresh arterial blood in that which is supplied to the nerve-fibres and ganglion-cells of the brain.—The freshly-oxygenated blood cannot get into the brain-substance, because the effete venous blood does not go out of it in some cases; in others, because the brain-substance is œdematous.—Œdema of the cerebral substance and its consequences.—It compresses the cerebral capillaries and thus produces anæmia of the nerve-fibres and ganglion-cells.—Acute œdema of the brain occurring in Bright's disease.—The efficient cause of the congestive and serous forms of apoplexy a suddenly produced anæmia of the brain-substance, or stoppage in the supply of arterial blood to the nerve-fibres and ganglion-cells.—Cerebral hemorrhage suspends the functions of the brain, not from pressure of the extravasated blood on the brain-substance itself, but from the compression of the cerebral capillaries and consequent anæmia of the brain-substance which it produces.—Value of abnormal pulsation or throbbing of the carotids as a symptom.—Post-mortem appearances found in cases of cerebral anæmia produced by compression of the cerebral capillaries.—The author's experience concerning the anæmia of the brain-substance which is produced by depressed fracture of the skull.—In apoplectiform cerebral embolism the phenomena are obviously due to sudden stoppage of the blood-supply to the nerve-fibres and ganglion-cells of the brain.—Nervous apoplexy also due to anæmia of the cerebral substance.—Durham has shown that the brain is anæmic during sleep.—Mushet found the brain of an infant poisoned with laudanum very pale.—The author found the brain-substance very pale and anæmic in a young woman poisoned with opium or morphia.—Bedford Brown found chloroform-sleep to be attended with cerebral anæmia.—According to Trousseau, epileptic stupor is attended with anæmia of the great nervous centres, at the outset of the fit at least.—Ackerman has shown by experiments on animals that asphyxia is invariably accompanied by cerebral anæmia.—The efficient cause of apoplexy in all its forms concisely stated.—The anatomical lesions which give rise to apoplexy also briefly stated.

WHAT is the efficient cause of this disease? What is the lesion of the brain-substance which paralyzes the cerebral nerves and thus occasions the phenomena of apoplexy?

For a long time the doctrine has been generally accepted that apoplexy, or at least the congestive and hemorrhagic varieties of it, are occasioned by abnormal pressure on the brain, that is, by abnormal pressure exerted upon the ganglion-cells and nerve-filaments of the brain. It has been generally believed that, in cases of congestive apoplexy, this pressure results from over-distension or excessive hyperæmia of the cerebral blood-vessels, and that, in cases of hemorrhagic apoplexy, it results from extravasation of blood into the brain through the bursting of some of these vessels. This abnormal pressure, which has been supposed to paralyze the ganglion-cells and nerve-filaments of the cerebrum, and thus suspend its functions together with those of the cerebral nerves, or, in other words, produce the symptoms of apoplexy, is, in the first-mentioned cases, called *intra-vascular*, because it is exerted by the blood from the inside of the distended vessels, and in the last-mentioned cases *extra-vascular*, because it is exerted by the extravasated blood from the outside of the bursted vessel or vessels. There have been some writers, however, who long ago doubted whether " a sudden pressure upon the brain is necessary to produce the apoplectic state." For example, Heberden says: " Theory may teach, but will find some difficulty in proving, that apoplexies must arise from a compression of the brain, owing either to a distension of the blood-vessels, or to extravasated blood from their rupture, and that the energies of the nerves can be deadened by no other cause." (Vide *Commentaries on the History and Cause of Diseases.* Art. *Paralysis et Apoplexia*, p. 354. London, 1802.) Others, again, have found it difficult, in some cases of apoplexy, to account for its causation by the hypothesis mentioned above; for when the quantity of blood extravasated has not been larger, for instance, than a barley-corn, it is not easy to see how the phenomena of apoplexy have been produced by mere pressure from extravasated blood. (*Aitken.*) Still, as already stated, the opinion has generally prevailed that apoplexy is, for the most part, produced by some sudden pressure upon the brain, either intra-vascular or extra-vascular in situation and spontaneous in origin.

But something more than sudden and excessive hyperæmia of the cerebral blood-vessels is required in order to satisfactorily account for the phenomena of congestive apoplexy, since an enormous amount of intra-vascular pressure on the brain is often borne without producing an apoplectic state. For example: Children affected with whooping-cough may have so many fits of coughing in rapid succession that an intense degree of congestion of the head is thereby brought on; so much so, indeed, that they may have hemorrhage from the nose, that their face may remain persistently puffy, and ecchymoses may, in some cases, form beneath the eyelids. There can be no doubt about the brain participating in the congestion. The fit over, they remain for a while in a state of bewilderment, but do not exhibit the symptoms of apoplexy. (*Trousseau.*) Again, see what happens in a woman during labor As the child's head is passing through the inferior outlet of the pelvis and the external organs of generation, she often makes the most violent efforts. Her face becomes livid, her lips and eyelids swollen, her skin gets hot and bathed in sweat, and there can be no doubt but that the sinuses of the dura mater, and the whole substance of the brain, share in this congestion. (*Trousseau.*) Furthermore, acrobats, who go through many of their performances with their heads hanging downwards, never suffer from any thing like apoplectic stupor; and the drivers of beer-wagons, who all day long carry heavy burdens in the shape of barrels of lager-bier, get almost purple in the face, whilst the blood-vessels of the neck are turgid and look like knotted cords, are not subject to deprivation of consciousness or muscular power at the moment when they are exerting themselves the most. These facts conclusively prove that something more than purely physical congestion of the brain is required in order to produce the phenomena of apoplexy.

Here we may with propriety make a remark which has special importance in this connection, namely, not only is extreme cerebral congestion, as we have just shown, often unattended by any apoplectic phenomena whatever, but, on the other hand, the symptoms of apoplexy, that is, the sudden suspension of all the cerebral functions from paralysis of ganglion-cells and nerve-fibres, are not unfrequently produced by a condition of the brain which is exactly the reverse of hyperæmia or intra-vascular pressure, namely, by an anæmic or bloodless condition, as we see in certain cases of apoplectiform cerebral embolism where some cerebral arteries of considerable size have suddenly become plugged up with embolia and the corresponding part of the brain deprived of its supply of nutrient blood. It is much easier to believe that the explanation of the phenomena of congestive apoplexy which is generally accepted is not correct, than it is to believe that the increase of intra-vascular pressure on the brain produces exactly the same symptoms, on the one hand, as the withdrawal of all vascular tension in the same parts does, on the other.

Niemeyer has enunciated some new views in regard to the pathology of apoplexy, which have the merit of being philosophical, and, as I believe, also the greater merit of being substantially correct. He says: "The reference of the symptoms of [cerebral] paralysis to an increase of the intra-vascular pressure appears to me erroneous, for even in the severest forms of hyperæmia this pressure does not nearly approach the grade necessary to induce paralysis of a peripheral nerve. In support of the above view, it has been said that paralysis is also caused by the scarcely greater extra-vascular pressure of small extravasations on the brain; but I shall hereafter show that the apoplectic symptoms from cerebral hemorrhage do not depend on compression of the brain from the extravasation. It is much more probable that the symptoms of [cerebral] depression and paralysis arise because the requisite supply of arterial, oxygenated blood to the nerve-filaments and ganglion-cells of the brain is limited or entirely stopped in excessive cerebral hyperæmia. In congestive [or passive] hyperæmia the escape of venous blood from the brain is checked; and it is evident that, when the veins finally become filled to a certain point, no new arterial blood can enter the capillaries. It is often asserted that the symptoms of cerebral hyperæmia are very similar to or exactly identical with those of cerebral anæmia; this is true in regard to congestive [or passive] hyperæmia and anæmia, and the explanation of the correspondence is easy. In both cases the brain lacks its new supply of arterial blood. To explain the symptoms of [cerebral] paralysis occurring in fluxionary hyperæmia [determination of blood to the brain] also, we must take the hypothesis that, during its course, there is a secondary œdema of the brain, as a result of which we have capillary anæmia, a condition directly opposite to the original hyperæmia." (Vide *Niemeyer's Text-Book of Practical Medicine*, vol. ii. pp. 158, 159, 1st Am. ed.)

Concerning the secondary œdema of the brain mentioned above he says: "At all events, in cases where there can be no doubt of death having resulted from increased flow of blood to the head, or from its obstructed escape thence, on a section through the brain, its substance is often found very pale, and on its cut surface only a few small blood-drops ooze up. This circumstance, and the symptoms of paralysis occurring in the severer cases of cerebral hyperæmia, cause it to appear to me very probable that, when there is increased lateral pressure in the small arteries and veins of the brain, a transudation of serum from them into the perivascular spaces and interstices of the brain may very readily take place, and cause compression of the capillaries. It is only in yielding and distensible organs and tissues, which are not inclosed by firm envelopes, that any considerable œdema can coexist with a normal fulness of the capillaries. In all tissues inclosed by fascia or other firm capsules, œdema causes anæmia of the capillaries. If the size of the brain be not diminished by atrophy, and if the skull be closed, or, it remaining open, if the dura mater be tense, there is no doubt that a slight transudation of serum will suffice to completely compress the capillaries of the brain. It is true, we cannot be sure, from *post-mortem* examination, that there is such a secondary œdema; but the supposition that such is the case appears to us perfectly justifiable when a patient has died with the symptoms of cerebral paralysis, and if, on autopsy, we find that the very white hue of the brain-substance and the slight number of small blood-points appearing on its cut surface contrast strongly with the distension of the large vessels in the meninges." (*Ibid.*, p. 156, vol. ii.)

There is another important variety of œdema of the brain which is connected with uræmia. Concerning it Niemeyer says: "But, in spite of the experiments of Munk, I hold it to be unproved, and even improbable, that the acute œdema of the brain of Bright's disease should have an origin different from that of the œdema of other regions, or that it should be ascribable to an increase of pressure within the cerebral arteries. Moreover, it seems to me to be extravagant to endeavor to ascribe all cases of so-called uræmia to compression of the cerebral capillaries, and to anæmia of the brain. My position on this question is as follows: In chronic parenchymatous nephritis, various organs are subject to œdema, the precise cause of which is unknown. It is characteristic of this œdema, that it shifts its position. It may attack the lungs at any period, either early or late in the disease, sometimes causing death, and sometimes subsiding again after a short duration. In a manner precisely similar, and for the same unknown reason, the brain may become the seat of an acute or subacute œdema, to which many succumb, while in others the œdema changes its position, and the patients are restored to a state of tolerable comfort for a period of variable duration. Many cases of so-called uræmic intoxication, but by no means all, are the result of œdema of the brain, and consequently anæmia of the cerebral capillaries. We may infer that an attack of this kind depends upon such an œdema, and not upon blood-poisoning: 1. When the seizure takes the form of deep coma, with intercurrent eclamptic spasms. 2. When, at the time of its occurrence, the secretion of urine is normal or increased. 3. When the attack is accompanied by marked œdema of the face. 4. When the carotids pulsate strongly during the attack. As we shall see directly, this is a valuable but often ill-appreciated sign of repletion of the cranial space with blood, and of impediment to the exit of the blood from the same." (*Ibid.*, vol. ii. pp. 22.)

Thus we perceive that the efficient cause of congestive apoplexy, serous apoplexy, and, in many instances, of the apoplectiform coma of Bright's disease also, is in reality the same, namely, a suddenly-produced anæmic state of the capillaries of the brain, whereby the requisite supply of fresh, arterial, oxygenated blood to the nerve-filaments and ganglion-cells of the brain is more or less suddenly stopped, and, consequently, the functions of the brain itself more or less suddenly and completely suspended.

We shall now proceed to speak of the pathogeny of hemorrhagic apoplexy, and we shall adhere closely to the views of Niemeyer on this subject, because we believe that they are in the main correct and that they advance us a long step in our knowledge of cerebral pathology. As already stated, it has been generally supposed that, in cases of hemorrhagic apoplexy or apoplectiform cerebral hemorrhage, the symptoms of cerebral paralysis are due to pressure upon or bruising of the nerve-filaments and ganglion-cells of the brain produced by the extravasated blood. But it is evident that this extra-vascular pressure exerted by the effusion can never exceed that of the blood in the cerebral arteries; for, as soon as the pressure in the parts around the ruptured arteries becomes as great as that of the blood in these arteries no more blood can flow out of them. Now, from experiments which we can make on peripheral nerves by compressing them, there is no doubt that such a degree of pressure is quite insufficient to destroy the excitability of or paralyze the nerve-filaments. Besides, the following fact also tends to disprove the current explanation: If the symptoms of paralysis depended on the pressure to which the filaments of the brain are subjected in apoplexy, then blood-letting should remove these symptoms at once, not only in some, but in all cases of apoplexy, provided enough blood be drawn to lessen the pressure in the whole vascular system, especially in the arteries. Hyrtl speaks most decidedly against referring these apoplectic symptoms to pressure on the brain. He thinks, however, that the phenomena generally ascribed to "pressure on the brain" are due to a slight amount of concussion of the brain; but, apart from the fact that no such concussion of the brain occurs in non-traumatic cases, no anatomical changes referable to such a cause can be found, and therefore his explanation is unsatisfactory. Niemeyer refers the apoplectic symptoms to sudden compression of the cerebral capillaries, that is, to suddenly produced anæmia of the brain-substance. In almost all cases of cerebral hemorrhage where the amount of the extravasation is large, this cerebral anæmia may not only be recognized with certainty after death, but even during life it shows itself by a very important symptom, which is often falsely interpreted, namely, by a remarkable pulsation or throbbing of the arteries in the neck and temples. This symptom is generally regarded as a sign of increased tendency of blood to the head, although it really denotes that the flow of blood into the skull is obstructed; we may at any moment produce the same phenomena in the artery of the finger by tying a string tightly around the end of the finger. All diseases of the brain and its membranes which diminish the space within the skull enough to prevent the flow of blood onward from the afferent vessels or arteries through the capillaries to the veins, such, for example, as large extravasations of blood, copious effusions of serum, abundant exudations of inflammatory products, large tumors, etc., are accompanied by increased pulsation of the carotids. If we find this symptom when there is no hypertrophy of the left ventricle of the heart, nor corresponding pulsation in other arteries, it will, in doubtful cases, be a great aid to the diagnosis of some brain-disease encroaching on the cranial cavity. Again, if we regard the physical conditions attending cerebral hemorrhage, we see that anæmia, at all events arterial anæmia, of the brain can never result from rupture of the cerebral capillaries; for the escape of blood from the ruptured capillaries can only last till the tension of the contents of the skull equals that of the blood in the capillaries. In accordance with this, we find that the apoplectic stroke does not occur in cases of capillary hemorrhage of the brain. If, on the other hand, a cerebral artery be ruptured, and the bleeding does not cease from some other cause, the tension in the surrounding brain-substance resulting from the extravasation finally becomes as great as it is in the artery; and, as the tension is greater in the arteries than it is in the capillaries, the latter must become compressed and finally impassable for arterial blood. In accordance with this, we find that arterial hemorrhages of any considerable extent in the brain are apt to be accompanied by the symptoms of a more or less extensive paralysis. Now, if we analyze the symptoms of an apoplectic stroke, we find that, while it lasts, the functions of both sides of the cerebrum are abolished. These patients have no feeling nor sensibility even when the strongest peripheral irritants are applied; they cannot make the slightest voluntary motion, and consciousness is entirely lost. But, all parts of the brain through which acts indispensable to the maintenance of life, and especially the respiratory movements, are performed, retain their functional power. This occurs, apparently, because the falx cerebri protects the opposite hemisphere from compression of its capillaries by the extravasation, less than the tentorium protects the medulla oblongata. Effusions of blood below the tentorium, even when slight, are very dangerous, because in such cases the medulla oblongata is not thus protected, and its functions are readily destroyed by compression of its capillaries. Niemeyer does not attempt to decide whether or not the slowness of pulse, the diminished frequency of respiration, and the contraction of pupil, observed in apoplectic fits from extravasations above the tentorium, are due to increased excitement of the vagus and oculo-motor nerves as a result of pressure acting on them, but somewhat modified by the tentorium. (Vide *Niemeyer's Text-Book of Practical Medicine*, vol. ii. pp. 197, 198, 1st Am. ed.)

Concerning the post-mortem appearances which are found in cases where death has resulted from anæmia of the brain induced by compression of the capillaries as a consequence of some disease encroaching on the intra-cranial space, Niemeyer says: "The pressure exerted by large extravasations of blood and extensive tumors is so great, that not only the capillaries and finer arteries and veins of the brain-substance, but also the larger vessels of the meninges, that are subjected to this pressure, are compressed and bloodless. If the disease be in one of the large hemispheres, it becomes more prominent after the skull is opened, so that on the affected side the dura mater appears more tense than on the other side. If the dura mater be opened and turned back, we see that the surface of the diseased hemisphere is remarkably even, that there is little, if any, liquid in the subarachnoid space, that the convolutions are lower, the furrows shallower, and that the vessels of the pia mater are not so full as natural, or are quite empty. Lastly, also, on section through the brain, we cannot fail to see a decided difference between the two hemispheres in regard to their color, and as to the number of blood-points appearing on the cut surface. In those cases where the falx and tentorium have to a certain extent given way [or yielded] to the pressure propagated to them, the falx showing a convexity [or bulging] toward the healthy side, the tentorium being flattened, or, when the disease is in the posterior cranial fossa, being more strongly curved, it is certain that the capillaries are compressed in those portions of the brain where the disease encroaching on the [intra-cranial] space is located. But at the same time it is found that the anæmia does not remain limited to the part of the brain first affected, but extends to other portions subsequently, although to a less extent." (*Ibid.*, vol. ii. p. 180.[1])

The author has made some observations on the living subject which strongly corroborate Niemeyer's views concerning anæmia of the cerebral capillaries as a consequence of pressure on the brain from extravasated blood. In operating for depressed fracture of the skull, he has several times found the dura mater lacerated, and thus has had an opportunity to see the condition of the underlying brain-substance. On raising up or removing the sunken portion of bone, he has several times noticed the corresponding depression in the surface of the cerebrum, and has remarked the anæmic or bloodless appearance which it presented. He has seen this sunken surface of the brain slowly elevated to its normal position by the internal forces which cause the cerebral pulsations, and he has observed that as the capillary circulation became reëstablished, the anæmic hue disappeared, and the natural rosy color of the part was restored. He has also noticed, on pressing his finger upon the exposed brain-surface with moderate force, that the spot pressed upon became pitted and pale or exsanguinated, and that the natural appearance of the part was speedily recovered after withdrawing the finger.

From the foregoing arguments and observations, both clinical and postmortem, we have no doubt that, in cases of apoplexy produced by extravasation of blood into the brain, the phenomena are due not to pressure upon the nerve-fibres and ganglion-cells, but to compression of the cerebral capillaries, whereby a state of anæmia of these fibres and cells, with corresponding arrest of the cerebral functions, is produced.[1]

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[1] We can readily pardon Niemeyer for making the following remark, although it seems rather vain at first sight: "I have been much gratified by the steady advance made by the doctrine which I propounded long ago, that the symptoms of the so-called cerebral pressure, due to encroachment upon the cavity of the cranium, whether by a depression of the skull, hemorrhage, tumor, abscess, inflammatory exudation, or serous transudation, all depend upon an arrest or obstruction to the flow of blood [through the capillaries] to the ganglion-cells and nerve-fibres of the brain." (*Ibidem*, vol. ii. p. 32.)

Concerning the pathogeny of apoplectic cerebral embolism there also is no room for doubt. The cerebral paralysis that occurs in such cases is obviously due to the anæmia of the brain which is produced by the sudden plugging up or occlusion of the cerebral arteries that constitutes the accident. The nerve-fibres and ganglion-cells in the affected part of the brain suddenly become deprived of freshly-oxygenated, arterial, or nutrient blood, and their functions as suddenly cease.

It remains for us to speak here concerning the pathogeny of the only other distinct variety of cerebral apoplexy, according to our classification, namely, nervous apoplexy. Cases in which from strong mental emotions, such as terror, grief, and despair, there occurs, without lessening of the heart's action, sudden pallor of the countenance, and even loss of consciousness, with other symptoms of insufficient supply of blood to the cerebral ganglia, seem to indicate that anæmia of the brain may also be caused by abnormal innervation or spasmodic contraction of the cerebral blood-vessels. (*Niemeyer.*) Nervous apoplexy is, in all probability, an intense form of cerebral anæmia, which is suddenly produced by spasmodic contraction of the cerebral arteries, or rather, of all of the cerebral blood-vessels having a muscular coat. After describing the symptoms of a form of anæmia of the brain sometimes due to emotional causes, which comes on suddenly and quickly attains a high grade, Niemeyer says: "In most cases the patients come out of this fainting fit in a short time; in other cases, usually termed apoplexia nervosa, consciousness does not return, the swoon ends in death." (*Ibid.*, vol. ii. pp. 170, 172.)

To return again to the pathogeny of congestive apoplexy, concerning which there seems to be more room for doubt with regard to its dependence on cerebral anæmia, than there is with regard to the dependence of the other varieties of apoplexy on cerebral anæmia, the following remarks of Niemeyer possess considerable value: Since it is not merely the presence of blood in the vessels of the brain, but the presence of oxygenated arterial blood that is indispensable for the normal performance of its functions, it is evident that, even in cases where the absolute amount of blood in the brain is not diminished, but where its circulation and distribution are changed, so that only a small amount of blood enters the organ through the arteries, and but little escapes through the veins, the same symptoms must arise as in true anæmia.[1] And the experiments of Kussmaul and Tenner entirely confirm the experience of pathologists, that, in degeneration of the heart, from non-compensated valvular obstruction, and in other diseases impairing its action, there is an overloading of the veins at the expense of the arteries, and a retardation of the circulation, that is, the same symptoms as occur in anæmia of the brain. (*Ibidem*, vol. ii. p. 171.) The apoplectiform symptoms which are not unfrequently produced by sudden and excessive hyperæmia of the brain unmistakably depend on the supply of oxygen to the ganglion-cells and nerves-fibres of the brain being arrested, so that Trousseau's phrase "cerebral asphyxia," is not far out of the way, after all.

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[1] Dr. Clutterbuck referred the symptoms of apoplexy to impeded or interrupted circulation in the brain, leading to suspension of the sensorial functions, from pressure operating upon the blood-vessels, so as to hinder mechanically the passage of blood through them. (*Mushet.*) His views, however, do not seem to have attracted that degree of attention among his contemporaries which they really merited. Niemeyer appears not to have been aware of Clutterbuck's opinions.

Such observations as the following may afford us some assistance in forming an opinion as to what is the condition of the capillaries of the brain, in respect to congestion or anæmia, in cases of apoplectic coma.

Mr. Arthur E. Durham has amongst others deduced the following conclusions from an experimental inquiry into the physiology of sleep: 1. Pressure of distended veins upon the brain is not the cause of sleep, for during sleep the veins are not distended; and when they are, symptoms and appearances arise which differ from those that characterize sleep. 2. During sleep the brain is in a comparatively bloodless condition; and the blood in the encephalic vessels is not only diminished in quantity, but moves with diminished rapidity. (Vide *Guy's Hospital Reports*, vol. vi. p. 149; also *New Sydenham Soc. Year-Book*, 1860, p. 43.)

Mushet found in a child aged three months, poisoned by laudanum, that the brain presented a very pale, almost anæmic appearance. The ventricles were slightly moistened with serum, and there was about a drachm at the base of the brain. The infant was flabby and ill-nourished. (Vide *Treatise on Apoplexy*, (*Cerebral Hemorrhage*,) etc., p. 162.)

The author found, in a young woman, otherwise healthy, who died from opium-poisoning, the substance of the brain pale and anæmic. Her case will be related in Chapter IV. See Case XXIII.

Bedford Brown observed during the administration of chloroform to a boy, aged ten, whose brain was exposed by extensive fracture of the skull, that the substance of the brain became remarkably pale or anæmic and shrunken when under the full influence of the anæsthetic, which was "a combination of sulphuric either and chloroform," and that its natural color and appearance were restored as the influence of the anæsthetic passed away. (Vide *American Journal of the Medical Sciences*, Oct. 1860, pp. 398-403.)[1]

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[1] The functional activity of the brain can only be maintained by the circulation in it of arterial blood; and whether no blood at all passes through the cerebral arteries, or merely venous blood, the result is substantially the same, namely, suspension of the cerebral functions, or a state of cerebral paralysis.

Trousseau says: "At the outset of an epileptic fit, the great nervous centres and the medulla oblongata of an animal subjected to experiment become paler instead of presenting signs of congestion." (Vide *Lectures on Clinical Medicine*, vol. i. p. 34; New Sydenham Soc. translation.) We may here remark that the phenomena of an epileptic fit bear a strong resemblance to the phenomena of an apoplectic fit, excepting the convulsive movements which characterize the former.

Ackermann has made experiments on animals for the purpose of ascertaining what the condition of the cerebral circulation is in cases of asphyxia. He removed a portion of the skull and the corresponding part of the dura mater from rabbits, closing the aperture with a glass plate and collodion. After about twenty-four hours, asphyxia was induced by strangulation, submersion, compression of the thorax, etc., and the condition of the brain observed through the glass plate. Asphyxia was found to be invariably accompanied by cerebral anæmia, instead of cerebral congestion. (Vide *Arch. f. Path. Anat.* tom. xv. p. 401; also *New Sydenham Soc. Year-Book*, 1860, pp. 130, 131, and *ibid.* 1861, p. 201; also *Mushet*, p. 163.) It thus becomes evident that in many varieties of fatal coma the capillaries and substance of the brain are really anæmic instead of being congested, as generally supposed.

There is, however, still much need of additional observations, both clinical and post-mortem, to show precisely what the condition of the cerebral substance or its morphological constituents is in congestive apoplexy; but upon the whole we do not perceive any good reason for doubting that it is substantially a state of anæmia. Considerable additional information on this subject may be found in our chapter on Congestive and Serous Apoplexy, especially the latter part of it.

We are now prepared to answer the questions which were asked at the commencement of the chapter. The efficient cause of apoplexy is a sudden suspension of or failure in the supply of fresh, oxygenated, arterial blood to the nerve-fibres and ganglion-cells of the brain, or, in other words, it is cerebral anæmia of great intensity and suddenness of production. The anatomical lesion which gives rise to apoplexy is anæmia of the cerebral capillaries, due either to stagnation of effete venous blood, or to embolism of the cerebral arteries, or to great diminution in size of the capillaries themselves from spasmodic contraction, from pressure exerted by surrounding œdematous infiltration, and from compression effected by extravasated blood.

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[1] Dr. Bedford Brown remarks: "It is conceived by the profession that under the impression of chloroform the vascularity of the brain is increased; that absolute congestion takes place. The present case afforded an ample opportunity to determine this question definitely. Whenever the anæsthetic influence began to subside, the surface of the brain presented a florid and injected appearance. The hemorrhage increased, and the force of the pulsations became much greater. At these times, so great was the alternate heaving and bulging of the brain, that we were compelled to suspend operations until they were quieted by a repetition of the remedy. Then the pulsations would diminish, the cerebral surface recede within the opening of the skull, as if by collapse, the appearance of the organ becoming pale and shrunken, with a cessation of the bleeding. In fact, we were convinced that diminished vascularity of the brain was an invariable result of the impression of chloroform or ether. The changes above alluded to recurred sufficiently often, during the progress of the operation, in connection with the anæsthetic treatment, to satisfy us that there could be no mistake as to the cause and effect. Again, the changes began invariably to appear with the earliest influence of the remedial agent. As consciousness diminished, in a corresponding ratio there was diminution of vascularity, and as the mental powers returned the contrary would occur." (*Ibidem*, pp. 400, 401.)

CHAPTER III.

ON THE CAUSES OF APOPLEXY.

1. *Influence of advanced age in the production of this disease.*—Opinions of Hippocrates and Galen among the ancients, and Drs. Cheyne, Bright, Watson, Copland, Mushet, and Aitken among the moderns, on this point.—The author's statistical tables illustrating it, and what they show.—The circumstances favoring the disposition to apoplexy which exist in old age.—Infants also perish of this disease much oftener than is generally supposed.—2. *Influence of sex.*—Males more liable to it than females.—3. *Influence of the seasons in causing apoplexy.*—Opinions of Galen, Cullen, Maclachan, Falret, Rochoux, and Aitken on this subject.—The author's statistical tables illustrating it and what they prove.—Apoplexy occurs most frequently in early spring, and least frequently in early summer.—More fatal in cold weather generally than in warm weather.—April charged with the largest number of deaths from it, and June the smallest.—4. *Influence of alcoholic drinks in causing apoplexy.*—The abuse of alcoholic drinks has more to do in producing apoplexy than any other cause.—The reasons therefor stated.—Summary of the various organic lesions which are produced by alcoholic intemperance.—Changes in the brain most frequent and strongly marked.—It also induces vaso-motor paralysis of the cerebral blood-vessels.—5. *On overfeeding as a cause of apoplexy.*—High-living tends to produce this disease. 1st. by inducing a state of general plethora; 2d. by occasioning obesity.—6. *On the abuse of opium and other narcotics as a cause of apoplexy.*—Their use predisposes to the occurrence of cerebral hyperæmia.—Their long continued abuse occasions vaso-motor paralysis of the cerebral blood-vessels.—7. *On excessive brain-work as a cause of apoplexy.*—Clergymen, authors, and editors of daily newspapers especially liable to die of this disease.—Reasons therefor stated.—8. *On the emotional causes of apoplexy.*—They consist of mental shocks, strong passions, and powerful emotions of the mind.—9. *On hereditary predisposition* to apoplexy.—A special tendency to this disease is transmissible from parent to offspring.—The author's experience on this point.—10. *On the so-called apoplectic constitution or appearance of the patient.*—Rokitansky declares it to be mere hypothesis.—11. *On the relations of sleep to the occurrence of apoplexy.*—In considerably more than one half of the cases the attack begins during sleep.—12. *On certain diseases which predispose to the occurrence of apoplexy.*—They are some organic affections of the heart, some morbid states of the cerebral blood-vessels, some diseases of the kidneys, the gouty and rheumatic diathesis, suppressed hemorrhoids, amenorrhea, and especially "the turn or change of life."—13. *On concussion or commotion of the brain as a cause of apoplexy.*—One of the author's cases referred to in which this form of injury probably determined the occurrence of cerebral œdema, with compression of the cerebral capillaries, and anæmia of the nerve-fibres and ganglion-cells.—14. *On energetic expiratory efforts, such as straining at stool, as a cause of apoplexy.*—The etiology of apoplexy is generally a quite complex affair.—A number of different causes usually coöperate in its production.

1. *Influence of Age in the Production of Apoplexy.*—Hippocrates[1] enumerates apoplexy among the diseases incident to old age. (Vide *Aphorisms,* sec. iii. 31, p. 722 *Works of Hippocrates translated by Dr. Adams.* Sydenham Soc. ed. London, 1849.) In another place he says:

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[1] Hippocrates, born 460 B.C. and died 377 B.C.

"Persons are most subject to apoplexy between the ages of forty and sixty." (Vide *Aphorisms*, sec. vi. 57, *op. cit.* p. 761.) Galen,[1] following Hippocrates, holds apoplexy to be a disease of old age. (Vide *Claudii Galenii Opera Omnia*, vol. xvii. B. p. 649. Editionem curavit D. Carolus Gottlob Kühn. Lipsiæ, 1821–1833.) Dr. Cheyne met with most cases of apoplexy between fifty and seventy. Dr. Bright thought apoplectics were chiefly above forty. Dr. Watson says it is common after fifty. Dr. Copland considers it to occur most frequently in the advanced periods of life—between forty and seventy. Mushet asserts that it is met with most often after the fiftieth year. (Vide *Treatise on Apoplexy*, p. 65, London, 1866.) Dr. Aitken says apoplexy is not unfrequent between thirty and fifty, while after fifty it is one of the most frequent causes of death. (Vide *Science and Practice of Medicine*, vol. ii. p. 322, 2d Am. ed.) The following tables show that age exerts an important influence on the production of apoplexy:

Table showing the age, sex, etc., of those who died of Apoplexy in the city of New-York in three years, namely, 1867, 1868, 1869, *arranged in ten-year groups; compiled from the Annual Reports of the Metropolitan Board of Health by the author.*

		Under 10 years.	10 to 20.	20 to 30.	30 to 40.	40 to 50.	50 to 60.	60 to 70.	70 to 80.	80 to 90.	Over 90.	Total of each Sex.	Total of both Sexes.	Per cent of the whole number of deaths, that is, the deaths from all causes.
1867.	Males... .	13	4	19	22	25	31	28	17	6	....	165	274	1.17
	Females..	13	1	6	14	12	16	23	18	6	....	109		
1868.	Males....	12	3	9	35	32	46	42	35	3	....	217	366	1.43
	Females..	11	2	12	18	34	17	31	23	1	....	149		
1869.	Males....	12	3	3	37	45	44	44	20	7	1	216	398	1.58
	Females..	7	2	11	20	29	33	41	29	9	1	182		
	Totals....	68	15	60	146	177	187	209	142	32	2	1038	1038	1.40

Table showing the age and sex of infants and young children who died of Apoplexy in the city of New-York in three years, (1867, 1868, 1869;) *compiled from the Annual Reports of the Metropolitan Board of Health by the author.*

		Under 1 year.	1 to 2.	2 to 3.	3 to 4.	4 to 5.	5 to 10.	Total of each Sex.	Total of both Sexes.
1867.................. ...	Males, ...	6	4	1	1	......	1	13	26
	Females,..	9	3	......	......	1	......	13	
1868............	Males, ...	6	2	2	.. ...	1	1	12	23
	Females,..	9	2	......	......	......	.... .	11	
1869....................	Males, ...	9	2	1	......	......	......	12	19
	Females,..	4	1	......	......	1	1	7	
	Totals,....	43	14	4	1	3	3	68	68

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[1] Galenus, Claudius, flourished in the second century, (born A.D. 131, and died about A.D. 201.)

Before presenting the conclusions to which these tables lead us, it is our duty to say a few words concerning the degree of authority or credence to which their teachings are entitled. First, then, the cases embraced in them all belong to the category of cerebral apoplexy. Besides, through the praiseworthy action of the Metropolitan Board of Health, and through the zeal and intelligence of its officers and employees, very great pains were taken with the registration of these cases in common with that of others. Whenever the certificate of death was found to be incomplete, incorrect, or of doubtful import, it was returned to the physician making it for correction or further information. We therefore have good grounds for assuming that the returns of death upon which these tables are founded are approximately correct, or, at least, much more nearly right than any similar returns that have been published in New-York. In preparing these tables, the report for the year 1866 was not incorporated, because it embraces a period of only nine months, and the report for 1870 was not used because it had not been published at the time of this writing, September, 1871. Still the number of cases embraced is over 1000, (1038,) which seems to be sufficiently large to afford trustworthy results; at all events, it is much larger than any heretofore presented for the purpose of illustrating the etiology of apoplexy in this country.

Now, our tables very clearly show several things of importance. For example, they show that a considerably larger number of persons die of apoplexy between the ages of sixty and seventy than during any other decade of life; and that Cullen, Rochoux, and Mushet were right in stating that this disease is most common between sixty and seventy, especially when the diminished number of the population at this period is considered. According to our tables more than one fifth of the whole mortality from apoplexy occurs between sixty and seventy. The decade which comes next to it in fatality from this cause is from fifty to sixty, the one which stands next to the last is from forty to fifty, next to it from thirty to forty, and next to this comes the ten years between seventy and eighty. People may indeed die of apoplexy at any time of life, but before the age of thirty the mortality from this cause is comparatively small, as our tables clearly show. After thirty, however, the death-rate from apoplexy increases very rapidly until the age of seventy is reached, when it declines with equal or even greater speed. There are many circumstances which favor the disposition to apoplexy that exists in old age. At that period the capillary circulation becomes impaired in most regions, and thus the veins become filled with more than the normal quantity of blood, or they may be distended from congestion. The cerebral arteries also are often diseased; the aorta is often stiffened from atheromatous degeneration of its walls; the heart has frequently acquired an abnormal power, driving the blood with great violence and with increased momentum toward the brain, while the lungs have their functions so much impaired that the blood is only imperfectly oxygenated; and these things are all causes of congestion, and of tendency to rupture of the vessels of the brain. (*Aitken.*)

Our tables also show that infants perish of apoplexy much oftener than is generally supposed. Thus, sixty-eight children of less than ten years died of this disease during the period embraced in the reports. Of these forty-three, or almost two thirds, were less than one year old. We shall, however, consider this subject with the fulness which it deserves in the Chapter on Infantile Apoplexy.

Again, these tables show that the total number of deaths from apoplexy in the city of New-York for the years 1867, 1868, and 1869 amounted to the large sum of 1038, or 1.4 per cent of the whole number of deaths—that is, the deaths from all causes combined.

2. *Influence of Sex in the production of Apoplexy.*—Galen held that males are more liable to be attacked by this disease than females. (Vide *op. cit.* vol. v. p. 696.) Mushet says it occurs oftener in males than in females. (Vide *op. cit.* p. 65.) But Dr. Aitken states that both sexes are liable to this affection and in nearly equal proportion. (Vide *op. cit.* vol. ii. p. 322.) He is, however, doubtless mistaken; for not only is the weight of authority against him, but our tables show that while 598 males died of apoplexy, only 440 females died of this disease during the same period. Thus, we find that the deaths among males from this cause exceeded the number of deaths among females by 158, which is certainly a very striking difference, as it amounts to 15.23 per cent of the whole number of deaths from apoplexy. Our tables also show that the mortality from this disease among infants and young children was considerably greater for males than for females, since 37 of the former and 31 of the latter are said to have perished from this cause.

3. *Influence of the Seasons in the production of Apoplexy.*—Among the ancient authors Galen, of his own motion, as well as when commenting on Hippocrates and agreeing with him, enumerates apoplexy among the diseases of the winter season. (Vide *op. cit.* vol. v. p. 694, and vol. xvi. p. 27.) Cullen believed persons to be most liable to attack from it in the spring. Dr. Maclachan considers apoplectic effusion to be greatly more frequent in winter than in summer, which agrees with the researches of M. Falret, who believed hemorrhage of the brain in Paris to be more common in the winter than in the summer and spring; but this does not accord with the experience of M. Rochoux, whose cases (69) were almost equally distributed among the seasons. (Vide *Mushet on Apoplexy*, pp. 65, 66.) Dr. Aitken very justly holds that extremes of temperature are powerful predisponents to apoplexy. In summer, the

fluids of the body tend to produce turgidity of the vessels in some constitutions and the tone of the capillaries is also impaired; while in winter the cold drives the blood from the periphery of the body to its central organs, and consequently to the brain. Sudden and great vicissitudes of the weather, as they rapidly exhaust the nervous power, are more frequently fatal than the uniform continuance of its extremes. (Vide *op. cit.* p. 321.) The following tables briefly set forth the relative influence of the different seasons in producing apoplexy:

Table showing the number of deaths from Apoplexy which occurred in each month from Jan. 1st, 1858, to Dec. 31st, 1865, (eight years,) in the City of New-York; compiled from the Annual Reports of the City Inspector by the author.

Months.	1858.	1859.	1860.	1861.	1862.	1863.	1864.	1865.	M'ly Totals.	Months.
January......	11	26	37	24	29	30	38	40	235	January.
February.....	19	17	23	29	30	31	25	27	201	February.
March........	24	28	30	26	33	28	28	28	225	March.
April.........	27	34	21	26	35	34	38	37	252	April.
May..........	13	21	15	24	27	23	16	37	176	May.
June.........	19	22	1	22	27	24	36	22	173	June.
July..........	8	22	28	28	10	28	32	30	186	July.
August.......	20	27	22	17	24	46	49	20	225	August.
September....	16	30	29	22	19	26	26	32	200	September.
October......	24	27	23	27	23	39	24	18	205	October.
November....	21	21	19	28	11	43	28	41	212	November.
December ..	26	24	30	18	15	31	29	24	197	December.
Yearly Totals.	228	299	278	291	283	383	369	356	2487	Grand Total.

The total number of deaths from apoplexy in the city of New-York for the eight years above mentioned amounts to 2487, which gives a yearly average of 310, as we have already stated.

Tables showing the influence of the Seasons on the mortality from Apoplexy in the City of New-York during eight years, from 1858 to 1865, inclusive; compiled from the Annual Reports of the City Inspector by the author.

Season.	Month.	Aggregate number of Deaths.	Total for Season.	Season.	Month.	Aggregate number of Deaths.	Total for Season.
Summer.	June,	173	584	Winter.	December,	197	633
	July,	186			January,	235	
	August,	225			February,	201	
Autumn.	September,	200	617	Spring.	March,	225	653
	October,	205			April,	252	
	November,	212			May,	176	

ANOTHER TABLE.

Season.	Month.	Aggregate number of Deaths.	Total for six Months.	Season.	Month.	Aggregate number of Deaths.	Total for six Months.
The six months of warm weather in the City of New-York.	May,	176	1165	The six months of cold weather in the City of New-York.	Nov.	212	1322
	June,	173			Dec.	197	
	July,	186			Jan'ry,	235	
	August,	225			Feb'ry,	201	
	Sept'er,	200			March,	225	
	October	205			April,	252	

Excess of deaths from apoplexy during the cold half of the year for the above-mentioned period of eight years, 157, or 6.03 per cent of the whole number of deaths from apoplexy during this period.

These tables show that the mortality from apoplexy in the climate of New-York is generally lowest during the summer months, that it increases in the autumn, that it increases still more in the winter, and that it becomes greatest in the spring. Cullen, therefore, was right when he thought that people were more exposed to attacks of apoplexy in the spring than at any other season. The months and seasons when the variations in temperature are most frequent, sudden, and considerable at New-York are attended with the largest mortality from apoplexy. April stands charged with the greatest number, and June with the smallest. The mortality from apoplexy is about the same in the balmy months of May and June. In those two months the deaths from this cause were 349, while in the changeable and trying months of March and April they amounted to 477, that is, the mortality of the latter period exceeded that of the former by 128, which is, relatively speaking, a very large number. The sultry and depressing heat of August and the intense cold of January each cause a striking increase in the death-rate from apoplexy. The influence of the dog-days of August in this respect is even more conspicuous than that of the frosts of January.

4. *On the Influence of Alcoholic Drinks in the production of Apoplexy.*—According to the author's experience, the abuse of alcoholic drinks has much more to do with the production of apoplexy in New-York, than any other cause. Most of the cases which have come under his own observation, infants excepted, have occurred in persons who drank to excess. In about two thirds of the original cases of apoplexy related in the following pages the causation of the disease was obviously connected with alcoholic intemperance. Dr. Aitken justly observes that among the most frequent of the causes of apoplexy, especially in some constitutions, is an intemperate use of fermented liquors—a class of substances which powerfully excite, mainly by inducing a paralysis of function, that allows the evidences of

excitement to appear as the prominent phenomena. (*Anstie.*) Alcohol also acts directly on the heart and arteries, causing not only temporary energetic action of these organs, but likewise, in time, organic alterations in their structure. In the latter case the powers of the heart are often permanently augmented, while the coats of the arteries, thickened, thinned, or ulcerated, are weakened, and also have their elasticity greatly impaired; and thus the tendency to congestion of and hemorrhage into the brain becomes greatly increased by the prolonged abuse of alcoholic drinks.

The pernicious effects of the continuous use of alcoholic stimuli on the organs and tissues of the body have been deduced from a careful study of the morbid appearances, of a chronic kind, which have been found in the bodies of persons known to have lived intemperate lives, and who had perished suddenly from accidents, suicide or homicide, and while, apparently, in ordinary health and activity. The extent of the chronic changes in the various organs of these persons has been found to be far in excess of that which is found in other persons of similar age, but of temperate habits, suddenly cut off while apparently in average health and vigor. The cumulative effects of long-continued intemperance have been clearly proved by Dr. Ogston's observations; and the results of his post-mortem inspections, on the whole, support the conclusions which have been arrived at on theoretical grounds as to the injurious effects of alcohol when abused. The following statements contain a summary of these results: 1. The great nervous centres present the largest amount of morbid change, the abnormal appearances within the cranium extending over 92 per cent of those examined. By this observation the theoretical remarks of Leovèille, Craigie, and Carpenter are clearly established. 2. The lesions of the respiratory organs succeed in frequency those of the nervous centres, yielding a percentage of 63.24 of those examined. 3. The morbid changes in the liver are next in order of frequency, and are due to enlargement, granular degeneration, the nutmeg-like congestion, and, lastly, the fatty state. 4. Next to the lesions of the liver come those of the heart and large arteries. 5. Next are those of the kidneys. 6. Least frequent of all are morbid changes of a striking character in the alimentary canal. Two orders of changes may be observed to result from intemperance in the use of alcoholic drinks, namely, one set of long duration, or which at least must have taken some considerable time for their evolution and completion; another set of shorter duration, and which are probably more closely connected with the immediate symptoms which precede the fatal event. The lesions found in the cranium, the substance of the brain, the convolutions, the ventricles, all indicate the prolonged action of a morbific agent. The consequences of the prolonged action of alcoholic drinks on the cranial contents are induration of the cerebral and cerebellar substance in a large majority of cases, coincident with an increased amount of subarachnoid serum; while the steatomatous degeneration of the small arteries of the brain induces atrophy of the convolutions and œdema of the substance of the organ. (Vide *Aitken's Science and Practice of Medicine*, vol. i. pp. 772, 773, 2d Am. ed.) We shall have occasion to speak again of the anatomical lesions which are produced by intemperance when we come to relate our own cases of apoplexy. But the habit of drinking to excess, like that of opium-eating, tends powerfully to induce vaso-motor paralysis of the cerebral blood-vessels, with passive dilatation of their calibre, turgescence, and stagnation of their contents, stoppage in the supply of arterial or freshly oxygenated blood to the nerve-fibres and ganglion-cells of the brain, and consequent suspension of their functions, or a state of cerebral paralysis, which is called apoplexy when it occurs suddenly. It is in this way, according to the author's observation and experience, that alcoholic intemperance most frequently occasions apoplexy. In this connection read also the paragraph on "the abuse of opium and other narcotics as a cause of apoplexy," to be found a few pages hence. The very great and, it may justly be said, overshadowing importance of the abuse of alcoholic drinks, at least in this country, as a cause of apoplexy, must be our excuse for dwelling here so long on this topic.

5. *On Overfeeding as a Cause of Apoplexy.*—Persons addicted to much indulgence in rich and luxurious food are generally believed to be a great deal more liable to apoplectic attacks than those who live on simple fare. Very frequently the apoplectic fit occurs soon after a luxurious meal. Now, luxurious living assists in causing apoplexy in two distinct ways: (1.) It produces a state of habitual plethora or abnormal fulness of the blood-vessels, which is denoted by the full appearance which high livers generally present. Such people are often said to look as if just ready to burst with good-living. This state of habitual plethora means a state in which the vascular tension, the lateral pressure of blood in the arteries is also habitually increased, and this circumstance obviously favors the occurrence of rupture in cerebral arteries that are already weakened by fatty degeneration or by ampullary dilatation, and the consequent extravasation of blood into the brain, or hemorrhagic apoplexy.

(2.) Luxurious living tends to produce obesity, or undue accumulation of fat in the connective tissue. It also tends, especially after long continuance, to produce fatty degeneration of such internal organs as the liver, kidneys, and minute blood-vessels, especially when joined to want of exercise and indolent habits. Most of the animals in our menageries die of fatty degeneration resulting from a too liberal supply of respiratory food. It is not too much to say that the bad consequences of luxurious living are very much increased by want of exercise and indolent habits, and that these evils are still further and still more largely increased if, to the foregoing, indulgence in strong drink is added. Hartshorne justly observes that full-living, especially with alcoholic intemperance (even moderate) and indolent habits, predispose to apoplexy in a marked degree. It should never be forgotten by those who have to deal with such cases that persons who habitually over-indulge in the pleasures of the table, and in strong drink besides, are especially liable to the occurrence of vaso-mator paralysis of the cerebral vessels, and consequently to the occurrence of congestion. and stagnation of blood in the brain.

6. *On the Abuse of Opium and other Narcotics as a Cause of Apoplexy.*—In the next chapter we shall relate a case of this disease which occurred in a woman who had been an opium-eater for many years, but who had recently reformed. Dr. Aitken says: "The excessive use of narcotics, as opium or tobacco, is also supposed to predispose to congestion of the brain, and consequently to cerebral hemorrhage." (*Ibidem*, vol. ii. p. 320, 2d Am. ed.) Niemeyer speaks of "the continuous misuse of narcotics, a practice which has greatly increased since the introduction of subcutaneous injections of morphine," as a cause of cerebral hyperæmia, and therefore, of apoplexy. (Vide *op. cit.* vol. ii. p. 158.) Upon the etiology of this form of cerebral hyperæmia Niemeyer says: "Physiological experiments show that if the cervical portion of the sympathetic nerve be divided, the vessels on the corresponding side of the head become dilated. The cerebral vessels appear to be similarly affected by the [long-continued] use of spirituous liquors, by some poisons," for example, opium, etc. "We can hardly give any other explanation of these cases than that the walls of the [cerebral] vessels are paralyzed by the above influences, their calibre dilated, and their supply of blood consequently increased." (*Ibidem*, vol ii. p. 153.) In such cases the cerebral hyperæmia is said to result from paralysis of the vaso-motor nerves of the cerebral blood-vessels, or, in other words, it is said that the long-continued misuse of spirituous liquors, opium, etc., induces, first, paralysis of the vaso-motor nerves of the cerebral blood-vessels, and then hyperæmia of the brain. It is probable that in at least some of the instances where persons long addicted to the use of opium are found dead or die unexpectedly, with symptoms of coma and stertor, that death is occasioned not by an overdose of the drug, but by apoplexy, or sudden and excessive hyperæmia of the brain induced in the way mentioned above. The symptoms of *acute* poisoning, however, by alcohol opium, and other narcotics, do not depend at all, or only very slightly upon cerebral congestion. (*Niemeyer.*)

7. *On excessive Brain-Work as a Cause of Apoplexy.*—It has been observed that clergymen, authors, editors, and other persons who are compelled to perform severe mental labor, with but little respite, for long peri-

ods are especially liable to death from apoplexy. We very frequently hear or read of distinguished members of these professions being suddenly stricken down and dying of this disease. There is no doubt that excessive brain-work often induces cerebral hyperæmia, attended by characteristic phenomena, and that, when long-continued, it not unfrequently induces complete vaso-motor paralysis of the cerebral blood-vessels, with œdema or serous infiltration of the brain-substance, compression of the cerebral capillaries from this cause, and, in consequence thereof, anæmia of the ganglion-cells and nerve-fibres of the brain, with sudden paralysis of the cerebral nerves, and death from apoplexy. Niemeyer, in speaking of excessive mental activity as a cause of cerebral congestion, says: "I would particularly call attention to the last cause, as I have frequently seen dangerous hyperæmia of the brain after too prolonged mental labor, which resulted fatally from the occurrence of œdema." (*Ibidem*, vol. ii. p. 153.) The bad effects of excessive mental labor on the brain are all intensified by luxurious living, indolent habits, and especially by alcoholic intemperance. It is, perhaps, a fortunate circumstance that most persons can not labor with their minds to good advantage if, at the same time, they indulge in much eating and drinking; for if they could, excessive brain-work might prove to be much more destructive to human life than it now is.

8. *On the Emotional Causes of Apoplexy.*—Dr. Aitken says: "The powerful effects of *moral causes* in producing this fatal disorder are well known." (*Ibidem*, vol. ii. p. 321.) The moral causes of disease in general are all those things which injure the body from within through the actions of the sentient being itself. Among the moral causes of apoplexy may be enumerated, great mental shocks, strong passions, and powerful emotions of the mind. They are divisible into two distinct classes, the first of which embraces all the great emotions which rouse or elevate the spirits and send the blood coursing through the arteries to the brain with increased vigor and rapidity, such as anger or rage, sudden and extreme joy, as from the receipt of good news, etc.; the second embraces all the great mental shocks which depress the spirits and suddenly blanch the countenance, such as extreme disappointment, sudden fright, great terror, and hopeless despair, operating in cases where the heart's action is not essentially weakened. The moral causes belonging to the first of these classes are liable to produce apoplexy through a sudden hyperæmia of the brain, resulting from increased vascular activity; those belonging to the last through a sudden anæmia of the brain resulting from vaso-motor spasm or contraction of the cerebral blood-vessels. When cases belonging to either of these categories suddenly prove fatal and no lesion is revealed by necroscopy to account for death, the disease is said to be nervous apoplexy; and with much justice too, for its occurrence is mainly

due to the operation of purely nervous causes. We thus perceive why mental tranquillity is a matter of so much importance to those who are predisposed to apoplexy.

9. *On Hereditary Predisposition to Apoplexy.*—In some persons, at least, there exists a predisposition to attacks of apoplexy, due apparently to peculiarities of constitution or organization which have existed from birth, such, for example, as an abnormal weakness of the cerebral blood-vessels, or an undue excitability of the ganglionic nervous system. Now, as the personal peculiarities of form and feature are transmissible from parent to offspring, so, without doubt, the personal peculiarities of internal organization, mentioned above, are also transmissible from parent to offspring. Thus, a special proclivity to attacks of apoplexy is doubtless transmissible from parent to offspring. This special liability to the occurrence of apoplexy which is born with the child, is correctly said to be due to hereditary predisposition. In this sense apoplexy becomes or may be considered as a hereditary disease. It is in just the same sense that insanity is a hereditary disease.

The evidence of hereditary predisposition to apoplexy in some families is very strong. The author knows of one family wherein almost all the men and many of the women are said to have perished of apoplexy or the kindred disorders, paralysis and epilepsy, for three generations; and he has in mind another family, having now many branches, in which most of the men belonging to the original stock have died of this disease. Hence apoplexy is sometimes said to run in the blood of families. Among authors, Portal considered apoplexy to be so strongly hereditary, in many instances, that he made the hereditary a distinct variety of the disease.

10. *On the so-called Apoplectic Constitution, or Apoplectic Appearance.*—It is generally believed that plethoric, florid, full-faced, short-necked, big-bellied, fat, and indolent persons are especially liable to be attacked by apoplexy. Aitken says: "Those most liable to attack are the florid in complexion, of short-necked conformation, with prominent eyes, broad chests, and protuberant bellies, and sometimes enormously fat, especially if high livers, sedentary, and indolent. Many thin persons, with spare, long necks, however, die from apoplexy; but it is probable that in these cases the heart or large vessels have been diseased." (*Ibidem*, vol. ii. p. 323.) A perusal of the cases reported by Abercrombie and others shows that the florid and the pale, the fat and the lean, are numbered among the victims of this disease. Boerhaave insisted that short necks were always found in apoplectics. Ponsart affirmed that persons having small heads were especially disposed to apoplexy. Rochoux believed that there is no appreciable external sign which discloses a liability to this disease. Rokitansky declares that the so-called apoplectic constitution is mere hypothe-

sis. (*Mushet.*) Niemeyer says: "There is no such thing as an apoplectic constitution, indicated by a short neck and broad shoulders." (*Ibid.* vol. ii. p. 191, 1st Am. ed.) Thus we perceive that there is considerable ference of opinion among authors in regard to the so-called apoplectic constitution; but the weight of authority rests with Rochoux, Rokitansky, and Niemeyer. The author, however, has been led to believe from experience and reflection that the red-faced and plethoric are, in general, more subject to apoplexy than the pale-faced and meagre, because the former are more frequently addicted to those habits of eating and drinking which exert a very strong influence in the production of this disease, as we have already shown.

11. *On the Relations of Sleep to Apoplexy.*—After dinner or any protracted, luxurious meal, and during sleep, are the periods when apoplexy is most likely to occur. For example, Gendrin found that of 176 cases 97, or considerably more than one half, were attacked during sleep. The increased liability to the occurrence of apoplexy after a hearty meal is readily accounted for by the state of general plethora and increased vascular tension which are produced by stimulating food and drink, especially when they are taken in excessive or even in abundant quantity. But it is not so easy to account for the remarkable liability to the occurrence of apoplexy during sleep. It is, in all probability, not due to any cerebral congestion which the act of sleeping *per se* may induce, for Mr. Durham has conclusively shown that the brain naturally becomes anæmic during sleep. Besides, it is well known that many forms of cerebral congestion are attended with extreme wakefulness. To what then is the remarkable liability to the occurrence of apoplexy which exists during sleep to be ascribed? The answer is not clear. We know, however, that during this period the heart contracts with lessened force, and that the energy or vigor of the ganglionic nervous system appears to be temporarily diminished or depressed; and it may be owing to this circumstance that vaso-motor paralysis of the already diseased blood-vessels of the brain, and death from apoplexy, are more likely to occur in the sleeping than in the waking hours.

12. *On certain Diseases which predispose to the Occurrence of Apoplexy, and may therefore be considered as predisposing Causes of it.*—There are several diseases which more or less strongly predispose their victims to apoplectic seizures. We will enumerate the most important of them.

1. The following organic affections of the heart: *a.* Hypertrophy of the left ventricle, for it causes the blood to be sent to the brain with abnormal force, and likewise increases the tension or lateral pressure of the blood in the cerebral arteries. This affection of the heart sooner or later produces dizziness, sopor, muscæ volitantes, and other symptoms of cerebral conges-

tion. *b.* Non-compensated vavular disease of the ventricles, in consequence of which the natural current of the circulation is obstructed, the blood accumulates in the venous system, and the brain, in common with other organs, becomes congested. In non-compensated valvular disease of the left ventricle, the whole amount of blood in the vessels of the brain and its membranes is not increased, it is true, for, while the veins are overfilled, the arteries are less full; but the overfilling of the veins obstructs the flow of blood from the capillaries, which induces capillary hyperæmia and stagnation. The disturbance caused by valvular disease of the right heart is far greater than that which is caused by valvular disease of the left heart, for, in the former case, not only is the escape of venous blood from the brain impeded, but the entire amount of blood in the skull is increased. (Vide *Niemeyer's Text-Book of Pract. Med.* vol. ii. p. 154, 1st Am. ed.) *c.* The fibrinous deposits, which form on the valves of the left heart in cases of endocarditis, and constitute in the end the so-called vegetations may be broken off and carried away in the current of the blood to the cerebral arteries, and, by obstructing or plugging them, may deprive so large a portion of the brain of blood as to produce genuine apoplectic phenomena, or apoplectiform cerebral embolism. Thus, apart from hypertrophy, valvular disease of the heart predisposes to apoplexy in two distinct ways: First, by obstructing the circulation of the blood and thus inducing cerebral congestion, and, second, by allowing fibrinous concretions to form on them, which, on becoming detached, float away to the arteries of the brain and become cerebral embolia.

2. Disease of the cerebral blood-vessels is not unfrequently an important predisposing cause of apoplexy. On this subject Eulenberg, from his own experience, and a comprehensive review of that of others, concludes, (1.) that in a very large majority of cases, cerebral hemorrhage is due to degenerations of the cerebral arteries as its predisposing cause. In the smaller arteries these degenerations consist of fatty metamorphosis or simple atrophy, with the various forms of consecutive dilatation; while in the larger arteries of the brain there is endarteritis issuing in ossification, or fatty degeneration, or passive calcification. (2.) A not entirely rare cause of the rupture is true aneurism of the large cerebral arteries. (3.) Hypertrophy of the left ventricle will only favor cerebral hemorrhage when it permanently increases the normal tension of the arterial system. This is not the case in the compensating hypertrophy of valvular disease of the heart. (4.) In about one seventh of all the cases of apoplexy, neither predisposing diseases of the heart nor of the blood-vessels could be demonstrated. (Vide *Virchow's Archives*, vol. xxiv. p. 329; also *New Sydenham Soc. Year-Book*, 1862, pp. 81, 82.)

When the coats of the ascending aorta are deprived of their elasticity by disease, it acts as a predisposing cause of apoplexy of some importance, for, in such cases, the force of the heart unchecked by this elasticity acts directly on the cerebral arteries, and thus tends more strongly to rupture them and occasion cerebral hemorrhage.

3. Disease of the kidneys constitutes a predisposing cause of apoplexy of great importance. From chronic renal disease there results a chronic state of imperfect depuration of the blood, or retention in it of urea and sometimes other toxic agents. From this chronic poisoning of the blood arises, first, general impairment of the functions of nutrition, then disorders of the capillary circulation in general, with retardation and more or less stagnation of blood in the whole capillary system—follow, which render increased effort on the part of the heart necessary in order to carry on the systemic circulation, and finally hypertrophy of the left ventricle ensues, with corresponding tendency to apoplectiform attacks. The primary disease is of the kidneys; the secondary of the blood-vessels and the heart.

But this is not the only nor even the principal way in which the chronic poisoning of the blood that attends Bright's disease brings on an attack of apoplexy, for it is very liable to produce a suddenly-occurring œdema of the connective tissue in various parts of the body; and when it gives rise to sudden œdema of the brain, it produces sudden compression and anæmia of the cerebral capillaries, with corresponding paralysis of the nerve-fibres and ganglion-cells of the brain, or a fit of apoplexy, as we have already shown. Thus we see that degenerative disease of the kidneys acts both as a predisposing and a determining cause of apoplexy—the former by impairing generally the function of nutrition, disturbing the capillary circulation, and thus inducing hypertrophy of the heart; the latter by giving rise to cerebral œdema.

Furthermore, the general impairment of the function of nutrition which results from the chronic blood-poisoning of Bright's disease causes the cerebral blood-vessels, and especially the minute ones, to become atrophied or weakened in structure, and therefore more ready to burst from increase of vascular tension, or to allow serous exudation to occur when the lateral pressure is not sufficient to occasion rupture. It also causes the innervation of the cerebral blood-vessels to become seriously deranged, so that death with the phenomena of apoplexy is not unfrequently produced through either a vaso-motor spasm or a vaso-motor paralysis of these vessels which has its origin remotely in renal disease. Thus chronic disease of the kidneys becomes a predisposing cause of vaso-motor paralysis or vaso-motor spasm of the cerebral blood-vessels: and this circumstance affords one reason why the subjects of Bright's disease are so often struck down suddenly with apoplectic symptoms from the operation of intercurrent or accidental and apparently trivial causes. Thus we are able to understand how it is that persons who have never shown any symptoms of renal disease may suddenly die with symptoms of apoplexy, and on making a post-mortem examination no lesion of importance be found except the shrunken, contracted, or so-called gouty kidney.

4. The gouty and rheumatic diathesis are without doubt in some instances causes of apoplexy. Many well-authenticated cases of apoplectiform gout and rheumatism are on record; and several cases belonging to this category will be related in the sequel. The connection of the gouty kidney with this disease has been mentioned above. Garrod enumerates apoplexy among the gouty affections of the brain. (Vide *Garrod on Gout and Rheumatic Gout*, p. 515, London, 1863.) Garrod also found the blood to be rich in uric acid in some attacks of epilepsy, paralysis, and apoplexy occurring in persons not known to have gout. (*Ibidem*, p. 517.) Another writer of quite recent date says: "The gouty not unfrequently exhibit a marked tendency to apoplexy; which may either be dependent upon disease existing in the heart, which fails to supply the brain with a due quantity of blood and produces the serous form of the malady, or upon degeneration of the coats of the blood-vessels from atheromatous deposits, associated with an undue pressure upon them from an abdominal congestion and general plethora of the circulating system." (Vide *A Treatise on Gout, Rheumatism*, etc., by *Peter Hood*, London, 1871.) But the poison which circulates in the blood through the system in cases of gout and rheumatism appears also to be capable of disturbing the innervation of the cerebral blood-vessels in such a way as to suddenly produce vaso-motor paralysis of those vessels and an apoplectic fit, in such persons as happen to be predisposed to the occurrence of this neurosis.

While describing the different ways in which gout terminates fatally Dr. Gairdner says: "One of these is apoplexy. The same disposition to the deposit of earthy matter, which is so manifest in the articulations of the limbs, is equally evident in the arteries and veins. Their friable coats can no longer bear even the diminished impetus of the blood. But, though the heart is now acting with less energy, the pressure of the circulating blood is, for various reasons, oftentimes greater than before. The limbs and moving powers, it is true, have diminished in bulk, but the belly is more protuberant. Fat has accumulated around the great viscera, which, joined to the congested state of these organs themselves, and the impeded circulation through them, forces an unnatural flow towards the head and brain. The records of medicine are full of examples of apoplexy consequent on and arising from gout." (Vide *Dr. William Gairdner's Treatise on Gout, its History, Causes, and Cure*, p. 31, London, 1854.)

Besides the foregoing, many other diseases or pathological states of the body predispose to the occurrence of apoplexy, as, for example, mania, epilepsy, suppressed hemorrhoids, amenorrhœa, and especially "the turn or change of life." (*Aitken.*)

13. *Concussion or commotion of the brain*, even when moderate in degree, as, for example, when it is produced by the simple act of falling down, may determine the occurrence or excite an attack of apoplexy in a person already predisposed to the disease ; as it did in a case which came under the author's observation, and will be found related as Case XI., and probably in some other instances that have come under his personal notice.* In Case XI., the fall which the patient sustained appears to have determined the occurrence of œdema of the brain-substance, with consequent compression of its capillaries, anæmia of its nerve-fibres and ganglion-cells, and arrest of their functions, or an apoplectic state.

14. *Energetic expiratory efforts, such as occur in straining* at stool, cause cerebral hyperæmia by hindering the flow of blood from the veins of the neck into those of the thorax, and thus may induce an attack of apoplexy in any person who is predisposed from age, or disease, or intemperate and luxurious living, to its occurrence. For this reason, apoplectics are not unfrequently found lying insensible or dead in privies and water-closets where they have been attacked while attending to the calls of nature.

But the causation of apoplexy is not so simple a matter as a perusal of the foregoing pages might lead one to suppose if he is not well acquainted with the subject. It is in reality quite complex, that is, many causes usually coöperate in the production of individual cases of this disease. The great importance of the subject must be our apology for having discussed the causation of apoplexy at so considerable a length as we have done. This disease proves so very fatal to those who are attacked by it, that in order to stay its ravages we are in great measure restricted to preventing its occurrence, which we cannot do unless we have a thorough knowledge of its etiology.

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* On this point see Cases XXVII. and XXVIII., and the comments on both of them.

CHAPTER IV.

ON THE SO-CALLED CONGESTIVE AND SEROUS FORMS OF APOPLEXY.—THEIR CLINICAL HISTORY AND POST-MORTEM LESIONS.

Definition of these terms.—They are synonymous with simple apoplexy.—These varieties of apoplexy are closely allied to each other.—Generally hyperæmia of the vessels and œdema of the membranes of the brain do not coexist unless atrophy of the brain also is present.—*Case* I. Congestive apoplexy occurring in a young woman during menstruation; death in four hours; autopsy; excessive hyperæmia of the cerebral vessels the principal lesion; powerful emotions of her mind the exciting cause; pulmonary emphysema the predisposing cause.—*Case* II. Congestive apoplexy occurring in a female of intemperate habits; she quickly died; autopsy; strongly marked cerebral hyperæmia; alcoholic intemperance and the alcoholic lesions of the brain the predisposing causes, and great anger or rage the exciting cause of the attack; it is also probable that the latter induced sudden paralysis of the vaso-motor nerves of the cerebral blood-vessels, with dilation of their bore and stagnation of their contents.—*Case* III. Congestive apoplexy, resulting from chronic alcoholic intemperance; sudden death; autopsy; undoubted evidences of chronic hyperæmia and atrophy of the brain; chronic venous congestion of the organs generally; paralysis of the vaso-motor nerves of the cerebral blood-vessels also probably occurred.—*Case* IV. Apoplexy both congestive and serous, the result of intemperance and gluttony; death; autopsy; the so-called apoplectic configuration; well-marked œdema of the meninges and probably of the brain-substance also; hyperæmia of the large vessels of the brain and its membranes likewise present.—*Case* V. Found dead; autopsy; apoplexy both congestive and serous; habits temperate; senile atrophy of the brain present; the serous and congestive forms of apoplexy closely allied.—*Case* VI. Serous apoplexy occurring without warning in an intemperate subject, while engaged at his work; death in about fifteen minutes; autopsy; copious œdema of the substance of the brain and its membranes; post-mortem signs of cerebral œdema; cerebral atrophy, softening and anæmia of the cerebral capillaries; when did this œdema of the brain occur? In all probability partly before and partly after death.—*Case* VII. Serous apoplexy caused by alcoholic intemperance; venesection employed without benefit; death in ten minutes; autopsy; copious œdema of the meninges; brain atrophied, its substance indurated, and its blood-vessels dilated; how the abuse of liquor produces apoplexy; typical form of the toper's stomach described.—*Case* VIII. A middle-aged woman of intemperate habits found sitting on a chair dead; autopsy; found the lesions which characterize both serous and congestive apoplexy, together with those belonging to chronic alcoholism.—*Case* IX. A man of 58 found dead in bed; autopsy; found the lesions belonging to serous and congestive apoplexy; copious effusion of serum beneath the arachnoid and in the ventricles; brain atrophied, its blood-vessels dilated; walls of the lateral ventricles softened; cause of the disease probably high living; he did not drink to excess; plethora and chronic hyperæmia of the whole venous system.—*Case* X. Chronic alcoholism; sudden death in the cell of a police prison; autopsy; the man was committed on a charge of intoxication, but he died of disease; it was not a case of apoplexy; the coma came on too slowly for that; found the various organic lesions which are produced by intemperate habits.—*Case* XI. Chronic alcoholism; sudden death from the occurrence of congestive and serous apoplexy; autopsy; copious œdema of the meninges; vessels of the brain hyperæmic and dilated; substance of the brain atrophied and indurated; induration of the cerebral substance is often met with in old topers; Percy's experiments and what they prove.—*Case* XII. Found dead in bed, from apoplexy as it was supposed; habits not bad; autopsy; slight hyperæmia, but no œdema of the brain or its membranes; reasons for believing that he died of apoplexy given; brain not atrophied, therefore the congestion was transient.—*Case* XIII. Sudden insensibility attended by convulsive movements; death from coma a few hours afterwards; autopsy; found many of the lesions which characterize chronic alcoholism; the disease was epilepsy and not apoplexy.—*Case* XIV. Sudden death from eclampsia, occasioned probably by uræmia; autopsy; found Bright's disease of the kidneys, etc.; constitutional syphilis.—*Case* XV. Chronic disease of the kidneys without anasarca; uræmic convulsions; death; autopsy; albumen found in post-mortem urine, etc.; kidneys large, flabby, and congested.—*Case* XVI. Sudden death from softening of the brain attended by coma and convulsions; autopsy.—The last four cases have been related for the purpose of comparing their symptoms and post-mortem lesions with those of apoplexy.—*Case* XVII. Found dead in bed; cause thereof apoplexy, in all probability; autopsy; heart hypertrophied and dilated; mitral valves insufficient; brain presented a natural appearance; reasons why it is believed he died of apoplexy; spasmodic contraction of the cerebral blood-vessels, its nature and consequences.—*Case* XVIII. Senile bronchitis and emphysema of the lungs; sudden death from apoplexy; autopsy; senile atrophy and softening of the brain; post-mortem lesions incident to old age.—*Case* XIX. Sudden death from congestive apoplexy; no autopsy allowed.—*Case* XX. Congestive apoplexy; fatal in twenty hours; no autopsy; detailed account of symptoms and treatment.—*Case* XXI. Congestive apoplexy occurring in a woman of about fifty; recovery; symptoms and treatment fully related.—*Case* XXII. Congestive apoplexy with symptoms of hemiplegia occurring in a man of about sixty; recovery; symptoms and treatment also fully detailed.—Summary statement of the leading points in these cases of simple apoplexy, and the conclusions which they warrant.—*Case* XXIII. Suicidal poisoning from opium or morphia; death; autopsy; substance of brain pale and anæmic; pupils found to be dilated after death.

Definition.—The term *congestive* is applied to those cases of apoplexy in which the phenomena are due to hyperæmia of the brain, and the term *serous* to the cases in which the phenomena are produced by œdema of, or effusion of serum into the perivascular spaces of the brain-substance and the subarachnoid connective tissue. These terms, however, are used conjointly when the apoplectic phenomena are induced by the combined operation of cerebral hyperæmia, and cerebral œdema, as, indeed, happens in a large number of instances. They are also synonymous with the term *simple* apoplexy.

These two varieties of apoplexy are placed together in the same group, because they are closely allied to each other in respect to causation, phenomena, and consequences. They are not grouped together here merely for convenience of description, etc., for they really belong to one and the same class. If we say that they constitute two separate and distinct classes, we are effectually stopped by this practical difficulty, with regard to very many of the cases that come under our notice, namely, we cannot tell to which variety they actually belong.* But this point will be more fully shown by the history of some of the cases which we are about to relate.

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* "It must be remembered that types of apoplexy more abound in books than in practice." (*Mushet.*)

For a time the fact was ignored that on post-mortem examination the blood-vessels within the cranium were sometimes found distended and at others empty, as it was supposed that the amount of blood contained in the closed cranium of an adult could neither increase nor diminish, but was a constant quantity; and that anæmia or hyperæmia of these vessels was admissible only when the brain-substance was increased or diminished in quantity, that is, when there was hypertrophy or atrophy of the brain. This view was based on the following reasoning: The brain is not compressible, at least not by the pressure to which it is subjected from the contents of the blood-vessels; and it is surrounded by walls which do not expand; consequently only the same quantity of blood can enter the skull as passes out of it, and, conversely, only as much blood can pass out of the skull as enters it. This reasoning is false, as it starts with the supposition that the contents of the cranium consist only of the membranes of the brain, the brain-substance, and the blood-vessels with their contents; it leaves the cerebro-spinal fluid out of consideration. This, which is a simple transudation, can rapidly increase or diminish, and can at least partly pass into the spinal canal, which is not entirely inclosed by rigid walls. In almost all autopsies it may be seen that the amount of blood contained in the vessels and the amount of cerebro-spinal fluid are in an inverse proportion; that a distention of the vessels of the meninges is accompanied by a decrease of arachnoid fluid, and, conversely, that, when the vessels are less full, the meshes of the textus cellulosus subarachnoidealis contain a greater amount of serum. Only when the brain is atrophied do we find œdema of the membranes associated with overfilling of the vessels; and only when an effusion of blood, a tumor, or a collection of fluid in the ventricles has encroached on the intra-cranial space, do we find, along with anæmia of the cerebral blood-vessels, dryness of the membranes, and disappearance of the sulci between the cerebral convolutions. (Vide *Niemeyer's Text-Book of Pract. Med.* vol. ii. pp. 151, 152, 1st Am. ed.) With these introductory remarks we shall proceed to consider the clinical and post-mortem history of simple apoplexy.

Case I.

Congestive apoplexy occurring in a young woman during menstruation; exciting cause, strong mental emotion; death in four hours; autopsy.

Mrs. Waldron, a quadroon, aged 22, married, and the mother of two children, the youngest of which was three years old, was suddenly seized with an apoplectic fit a little before 9 o'clock on the evening of July 3d, in consequence of anger and shame occasioned by the discovery to her husband of a note containing the proof of her infidelity to him. Dr. Williams was immediately called. He found her sitting in a chair supported by three women. She was totally unconscious, breathing stertorously, and had slight spasms of the upper extremities, while the lower were unaffected. Her pupils were not contracted; pulse slow and rather weak. There being a suspicion that she had taken laudanum, an emetic of zinci-sulph. was administered, which failed to operate with sufficient promptitude, and the stomach-pump was then employed. The contents of the stomach were entirely free from the smell of opium or laudanum. The stomach was carefully washed out with warm water. There was no proof whatever that she had taken poison. She was menstruating. She continued in a state of profound stupor, having tonic spasm of the lower jaw alone, and died comatose about 12½ o'clock on the same night. She was said to be very passionate, and frequently before when aroused by anger had fallen into a state of insensibility.

Autopsy, by the author, eleven hours after death. Cadaver plump, in good preservation, and rigor mortis strong. Pupils somewhat dilated, both alike.

Head.—On dividing the scalp, blood in more than usual quantity flowed away. While sawing the skull-cap off considerable blood ran out, and after it was removed and the dura mater turned back, not less than ten or twelve ounces of blood, including that already mentioned, escaped. This blood was poured out from the sinuses of the dura mater and from the vessels of the brain. The skull was thick, the glandulæ Pacchioni larger and more numerous that usual. The visceral arachnoid exhibited some opacity about the vertex. The subarachnoid serum was rather more abundant than usual about the vertex, but not elsewhere. The vessels of the pia mater were everywhere gorged with blood. On slicing down the brain, the surface of each section was smeared over with blood, which, escaping from the divided blood-vessels, or puncta vasculosa, followed the knife. The brain-substance was normal in color and consistence. The lateral and other ventricles were nearly empty. No clots of blood were found, though careful search was made.

Chest.—On opening the thorax, it was observed that the lungs did not collapse, but rather tended to protrude from that cavity. They were pale in color, (their hue resembled that of a new-born child's lungs just after respiration has been fully established,) light in respect to weight, and very large in volume. Their increased size was due to a pretty uniform dilatation of the pulmonary air-cells, (vesicular emphysema.) On incising them, the cut surface was found to be quite dry, except a little pale, frothy serum, (slight œdema pulmonum.) The pulmonary tissue had a woolly feel. The bronchial tubes were natural. Each pleural cavity contained four or six ounces of serum having a slightly reddish tinge. The heart was somewhat hypertrophied, rather fatty and firmly contracted. The mitral valve, in both segments alike, was thickened by interstitial deposits of semi-cartilaginous consistence. The aortic valves were slightly thickened in the same way. Owing to persistent contraction, the cavity of the left ventricle was small and its walls thick; that is, there was false concentric hypertrophy, while the organ was really larger that natural. There were no heart-clots. The foramen ovale was closed.

Abdomen.—The liver, spleen, and kidneys appeared to be perfectly normal. The stomach was well contracted and nearly empty. Its mucous membrane was somewhat reddened, and thickly coated with mucus having a lemon-yellow tinge. The intestines were not opened, but externally they presented a normal appearance.

Pelvis.—The uterus was large, having a vertical diameter of 3½ inches, and a breadth of 2 inches, and presented a congested appearance. The os uteri was large, patulous, and exhibited menstrual blood streaming out of it. The substance of the organ was normal. Its cavity was lined with menstrual blood. The ovaries presented a congested appearance, but the left was reddened more than the right. The left ovary contained a fresh corpus menstruale, and its peritoneal investment exhibited a minute laceration through which it is probable that an ovum had recently escaped. The left Fallopian tube also was more congested than the right. The right ovary contained an old corpus menstruale. It was as large as a corpus luteum, firm in feel, and consisted of a shut sack filled with grayish-white, firm, fibrinous, or lardaceous material. Its walls were yellowish in color corrugated, and of varying thickness.

Comments.—Was this woman poisoned by opium? After mature deliberation, we unhesitatingly answer no. We are quite positive in this opinion, firstly, because there was not a particle of evidence that she had taken opium or any other poison; secondly, because she did not sink gradually into coma, as she would have done if poisoned by opium, but, on the contrary, she became suddenly or instantly insensible; and thirdly, she did not exhibit any of the phenomena which characterize poisoning by opium. Again, was this woman's fit an attack of epilepsy? To this question also we must reply in the negative, for she did not have general convulsions, she did not cry out when the fit began, she did not froth at the mouth nor bite her tongue, she did not void her urine nor fæces, and, finally, she did not present any of the symptoms which characterize an epileptic fit. Was this a case of so-called cerebro-spinal meningitis? We answer no, because the attack was not attended by any pain in the head, or neck, or back, nor by any stiffness of the neck, nor by any suspicious symptoms whatever, that is, it came on entirely too suddenly for that disease, and because the autopsy revealed no evidence whatever that there had been inflammation of the membranes of the brain and spinal cord. There is no doubt then that this was purely a case of apoplexy, and that the attack had been brought on by powerful emotions of the mind.

The evidences of cerebral congestion which were revealed by the post-mortem examination of this subject are very clear and convincing. The quantity of blood which escaped from the vessels of the head, including both the superficial and the deep-seated ones, while the brain was being exposed and examined, was very large, we may with propriety say, enormous, being not less than ten or twelve fluid ounces. At the same time the vessels of the pia mater and the substance of the brain itself were still gorged with blood. The congestion of the latter was so strongly marked that the surface of a section was literally smeared over with blood which escaped from the divided vessels, instead of exhibiting simply the so-called puncta vasculosa. It should be remembered, however, that these evidences of ante-mortem cerebral congestion were more or less intensified by the remarkable fluidity of the post-mortem blood.

The autopsy also revealed extensive emphysema of the lungs. This lesion of the respiratory organs had doubtless a good deal to do with the causation of apoplexy in this instance. It lessened very much the capacity of the pulmonary vessels or circulation, and thus destroyed the balance between it and the systemic circulation. It induced a state of chronic overfulness of the veins of the body in general, and thus caused a state of chronic venous hyperæmia of the brain in common with other parts. Now, whenever the action of the heart, and consequently the movement of the blood, became much quickened in this case from the operation of any cause, the congestion and stasis of blood in the cerebral vessels became correspondingly increased, the arterial blood failed to enter because the venous blood did not go out, and at last the nerve-fibres and ganglion-cells of the brain were in this way deprived of their supply of arterial or freshly oxygenated blood, their functions were suddenly arrested, and a state of insensibility or apoplexy produced. Thus the pulmonary emphysema acted as the predisposing, and the emotions of her mind as the exciting cause of the apoplectic fit. Thus we can readily understand how the pulmonary emphysema was also at the bottom of those other fits, supposed to have been hysterical, to which she is said to have been subject whenever she got angry. The emphysema of her lungs was also the cause of the hypertrophy of her heart. This organ had to contract with increased power in order to send the blood, not only through the capillaries of the lungs, but through the systemic capillaries also, because of the over-fulness and torpidity of the circulation in the veins throughout the body, and thus its growth was promoted until it attained an abnormal size.

The account of the autopsy in this case is also very interesting for a reason which is not immediately connected with the subject we are studying, namely, she was menstruating at the time of her decease, and the organs concerned in that process were not overlooked at the necroscopy. Thus, the left ovary was found to contain a fresh corpus menstruale, and the peritoneum investing it exhibited a minute laceration through which, in all probability, an ovum had recently escaped. The left Fallopian tube was also congested. The uterus, likewise, was large and congested. Its increase in size above that which belongs to the wombs of multipara in general was principally due to menstrual congestion, and would have subsided on the departure of the menstrual period. The sound condition of her liver, spleen, kidneys, and intestinal canal, except the gastric catarrh which had followed the use of the stomach-pump, denoted that her general health was pretty good when the apoplectic fit occurred.

Case II.

Congestive apoplexy occurring in a female of intemperate habits, excited by strong mental emotion; death very speedily produced; autopsy; acephalocysts in lungs and liver.

Mrs. Elizabeth W., aged about 30, married and the mother of five children, an Irish emigrant of the laboring class, had been landed but six weeks. On the 7th of November, she went to a "christening," where she took a good deal of spirituous drink. At 11½ o'clock P.M., she returned to her boarding-house, in company with her husband, in a state of semi-intoxication. Soon after getting there some hard words passed between herself and husband, and she became very angry. She then suddenly fell down upon the floor in a state of complete insensibility, and almost immediately expired. She did not have convulsive movements nor any other symptoms of epilepsy. She was reported to have been a very irritable, high-tempered woman, and subject to "fits" whenever she got in a towering passion. It was also stated that while in these fits she generally appeared to be completely insensible, that sometimes she had convulsive movements and frothed at the mouth, that she had these fits frequently, that they were supposed to be hysterical, that she was in the habit of drinking to excess whenever she could get a chance to do it, that her poverty prevented excessive indulgence of this kind from occurring very often, that her health was generally good, that she had suffered from cholera and typhus in the old country, but from no other acute disorder.

Autopsy, by the author, fifteen hours after death. The body was in good flesh, the abdomen was swelled from post-mortem tympanitis, and the rigor mortis was strong.

Head.—Scalp thick and congested; blood flowed freely away while dissecting it off. On turning the dura mater back, the vessels lying on the surface of the brain were seen to be strongly congested. The little twigs were beautifully injected. The glandulæ Pacchioni were pretty well developed, that is, they had a moderate size. The visceral arachnoid was generally hazy, besides which it exhibited pearly opacity in spots and streaks. There was but little subarachnoid serum. The substance of the brain contained considerably more than the normal quantity of blood, that is, it was congested. The little blood-vessels traversing it exhibited some dilatation, produced apparently by chronic congestion. The puncta vascuosa were more numerous and larger than usual. The ventricles contained a small quantity of pale serum. The color and consistence of the brain-substance were normal. About eight ounces of blood and serum either flowed out from the cranial cavity or collected in the occipital fossa during the examination of the brain. There were no coagula.

Thorax.—There were numerous old bridle-shaped adhesions of the pleura about the lower lobe of the right lung. The apex of the left lung was adherent. Both lungs contained more than the normal quantity of blood; also, some old concrete miliary tubercles, nearly black in color, scattered pretty uniformly through their parenchyma. There was an old cicatrix at the apex of the left lung. In the right lung we found three remarkable cysts. The largest one was situated in the posterior part of the lower lobe, and had the size and shape of a small lemon. The other two were situated in the anterior or free margin of this lung; both were pear-shaped; one was about an inch and three fourths in length by an inch in breadth, the other an inch and one fourth by three fourths of an inch. These cysts presented a whitish appearance externally like the sclerotica, and did not allow light to pass through them. We dissected out the smallest one entire and opened it. On making a puncture, a perfectly transparent serum spirted out, and a limpid fluid continued to flow until the cyst was nearly empty, when a whitish substance, bearing a strong resemblance to a very fine white sand, was discharged, and immediately sunk to the bottom of the watch-glass in which the fluid was collected. On laying this cyst open, its interior was found to be covered with the same granular matter. The microscope showed these granules to consist of *echinococci.* The interior of the cyst was not lined with epithelium. The wall of the cyst consisted of a thin membrane, on the outside of which were placed layers of connective tissue which had accumulated as the size of the cyst increased, and constituted at the time of examination the greatest part of the cyst-wall. The heart was natural; and the blood in the heart and great vessels fluid.

Abdomen.—The liver was enlarged, flabby, and nutmeggy. The edge of the right lobe was thick and rounded off. On cutting into the organ, its substance was found to be somewhat nutmeggy and probably fatty. The right lobe was also adherent to the diaphragm by firm, old, grayish-white, false membrane. In the left lobe we found a large, and flaccid, or somewhat shriveled, acephalocyst, which extended entirely through from the convex to the concave surface of this lobe. No other hydatids were found. The kidneys presented a finely mottled appearance, and their cortical substance was rather paler than natural, but no other abnormity was noted. They were considered to be fatty in an early stage. The stomach and intestines were much distended with gas. The stomach had a large size. Viewed externally, its veins on both the greater and lesser curvatures were full of blood, (congested,) and looked like blackened cords. It contained about eight ounces of food, (meat and potatoes,) but little affected by the digestive process; odor of spirits not present. Its mucous membrane was mammillated, thickened, softened, friable, and bore a pretty strong resemblance to thin, rotten leather in the last three respects. The mammillations had a whitish color. There were some small spots in the fundus that were reddened; but, save these, the gastric mucous membrane everywhere had a pale color. No abnormity observed in the intestines except the gaseous distention. Spleen normal. Uterus very large, and surrounded by old adhesions, the results of a peritoneal inflammation of no recent date. The muscular tissue of the body presented no abnormity.

Comments.—Was this a case of apoplexy? We answer yes; and it presented not only the phenomena of that disease, but the form of it which was called by the ancients *attonitus morbus*, by the moderns, *ictus fulminis*, and by the French, *coup de sang*, from the suddenness of the stroke and the rapidity with which it proved fatal.

What was the nature of the other "fits" to which she was said to have been subject whenever she got angry? Were they epileptic or hysterical? They had been pronounced hysterical by physicians whom she had consulted, and after careful inquiry the author found no good reason for adopting a different opinion. She had no aura, she did not bite her tongue, and she did not pass her urine while in these fits. The convulsive movements were, in all probability, either simulated or eclampsic in character.

This case presents a strong resemblance to the preceding in several important particulars: 1. The evidences of cerebral congestion which were revealed by the post-mortem examination were remarkably strong in both of them. 2. But little serous effusion was found beneath the arachnoid, or in the ventricles, in either of them. 3. In both subjects the apoplectic stroke was immediately induced by strong mental excitement, that is, by the passion of extreme anger or rage. 4. Each of them was rather young in years to become the victim of apoplexy.

The autopsy revealed a remarkable dilatation of the small vessels in the medullary substance of the brain, due apparently to chronic congestion. Her habits were intemperate. Over-stimulation with alcoholic drink, continued for a long time, had doubtless produced a state of chronic hyperæmia of the brain, with corresponding dilatation of the cerebral blood-vessels, and thus had predisposed her to be attacked by apoplexy. Besides, her kidneys were beginning to undergo a fatty degeneration, and probably were also commencing to fail in depurating the blood; and blood poisoned with effete matter circulating in the already congested brain would materially aid in predisposing to apoplexy. In this case the predisposing causes of the apoplectic fit were alcoholic intemperance, together with the chronic cerebral congestion and the fatty degeneration of the kidneys which it induced. The exciting cause was strong emotion of the mind in the shape of anger. It is also probable that the latter caused the vaso-motor nerves of the cerebral blood-vessels to suddenly become paralyzed, the vessels themselves expanded, their contents stagnant, the brain still more congested, its nerve-fibres and ganglion-cells deprived of freshly oxygenated blood, and their functions suppressed. Thus, the *ictus fulminis* occurred and proved quickly fatal.

The autopsy of this case presents some very interesting features besides the lesions pertaining to congestive apoplexy. For example, there was a so called tubercular disease in the lungs, of an old date, which had been permanently arrested; together with hydatids of considerable size in the right lung and liver, which had not, so far as known, announced their presence during life by any characteristic phenomena. Moreover, hydatid disease of the lungs and liver, whether announced by ante-mortem phenomena or not, is not of so common occurrence but that it always attracts attention when we get an opportunity to investigate its anatomical relations after death. The structure of the cysts themselves and the nature of their contents have therefore been carefully recorded. But instances in which the so-called tubercular disease of the lungs has been permanently arrested after reaching the stage of ulceration, although not unfrequently met with, are still more worthy of attention, because only a few years ago it was generally believed that in such cases recovery was impossible. In this case, there had been a so-called tubercular cavity in the upper part of the left lung, which had become healed, and left a cicatrix in the pulmonary tissue at the apex of this lung, together with strong adhesions of the pleura in the same locality.

CASE III.

Congestive apoplexy, resulting from chronic alcoholic intemperance; sudden death; autopsy.

J. D., aged 50, had been a hard drinker of spirituous liquors for more than ten years. He came home on the evening of Dec. 4th, rather the worse for liquor. He conversed with his family, however, and seemed to be as well as usual. But about eight o'clock, he suddenly fell out of the chair in which he was sitting down to the floor, and expired without complaint and without a struggle. He was notorious among his neighbors for being, almost constantly, either drunk or decidedly under the influence of spirituous liquor.

Autopsy, by the author, sixteen hours after death. Cadaver plump, adipose tissue abundant, rigor mortis rather feeble.

Head.—Dura mater adhered to skull more strongly than usual at the vertex. Glandulæ Pacchioni enlarged and constituting firm bonds of adhesion between the visceral arachnoid and the dura mater. The visceral arachnoid itself was found to be cloudy and opaque, presenting a more or less pearly appearance, in spots of variable size, at some of which there was a slight thickening. There was more serum than natural in the cavity that is, on the free surface of the arachnoid, which escaped on opening the dura mater. There was also a moderate quantity of serum lying in the meshes of the subarachnoid connective tissue and filling the furrows of the brain. The quantity of serum in the lateral ventricles was small. The brain and its membranes, particularly the pia mater, contained a good deal more than the normal quantity of blood, that is, they were strongly congested. On carefully slicing down the brain, the puncta vasculosa were found to be unusually numerous and large. The substance of the brain was normal in color, and fully as firm as usual in consistence. Thin slices of it presented something like a "cribriform" or sieve-like appearance from dilatation of the little vessels which permeate its medullary tissue.

Thorax.—Both lungs exhibited some vesicular emphysema, and the left lung contained more than the normal quantity of blood. The heart, particularly the left ventricle, was hypertrophied. There were abnormal deposits of adipose tissue on the external surface of the right ventricle and about the base of the organ. Its muscular tissue was paler than natural and flabby. No valvular disease worth mentioning was observed.

Abdomen.—The liver was found to be considerably enlarged, and to exhibit well-marked fatty degeneration in all its parts. The spleen also was considerably enlarged, apparently from congestion and stagnation of venous blood. The gastric veins were larger than natural. The stomach was above the normal size, and contained four or six ounces of whitish liquid which exhaled a faint odor of spirits. Its mucous membrane was reddish in color, and somewhat thicker and softer than natural, throughout its whole extent. There were some small, brown, hemorrhagic spots scattered over the fundus and along the greater curvature of this organ. The intestines were congested with venous blood. Condition of kidneys not mentioned in the notes; but they probably did not exhibit any change of importance, for if they had it would have been described.

Comments.—The apoplectiform phenomena presented by this case were very strongly marked. The subject was suddenly struck down as by a powerful blow. Without warning he was deprived of consciousness, and after breathing stertorously a few times, expired. No convulsive movements whatever occurred. According to the testimony of all the eye-witnesses, and they were several in number, "he died without a struggle."

His fit of apoplexy was occasioned by his habits of alcoholic intemperance. Over-stimulation with spirituous drinks, continued in his case for years, had produced a state of chronic overfulness in the veins of the body in general, together with a state of chronic congestion and atrophy of the brain, which was denoted by the greatly expanded condition of the cerebral blood-vessels, and by the marked degree of cerebral hyperæmia which, at the same time, was accompanied by œdema of the meninges. Hyperæmia of the brain and œdema of its membranes cannot coexist unless the volume of the brain itself is absolutely diminished, and such a diminution of the volume is only another name for atrophy of the brain. Thus, in this case chronic habits of alcoholic intemperance had produced a state of chronic venous congestion in every part of the body; and in the brain a state of atrophy in addition to the congestion. On this point Niemeyer says: "Where the hyperæmia is often repeated, atrophy of the brain and decided dilatation of the vessels result. The vessels of the meninges which are unmistakably dilated, run a tortuous course; on section through the brain, we may distinctly see the gaping mouths of the vessels; the dilatation may even be observed in the capillaries on microscopical examination. There is plenty of serum in the subarachnoid spaces, the brain-substance is moist and shining. This appearance, which is frequent in topers, is readily understood, if we remember that, in atrophy of the brain, the fluid contents of the skull must increase so as to fill the cavity." (Vide *op. cit.* vol. ii. p. 156.) In this case of general venous congestion resulting from chronic intemperance, the hyperæmia with stasis of the blood, and the œdema of the brain with atrophy thereof, increased until finally the nerve-fibres and ganglion-cells of the brain were no longer supplied with oxygenated blood, and then paralysis of all the cerebral functions, or apoplexy, immediately ensued. It may very well have happened in this case that a suddenly occurring œdema of the peri-vascular tissue in the brain-substance, by compressing the cerebral capillaries, in the way which we have pointed out while discussing the pathogeny of congestive apoplexy, unexpectedly occasioned anæmia of the brain-substance, and thus acted as the exciting cause of the apoplectic fit.

The account of the autopsy in this case is very interesting for the further reason that it presents us with the pathological anatomy of *alcoholismus chronicus.* The induration and atrophy of the brain-substance, the "cribriform" appearance which it presented on slicing, the fatty, pale, and flabby condition of the heart, the fatty degeneration of the liver, which was well marked and far advanced, the abnormally capacious stomach, the dilated gastric veins, the thickened and softened gastric mucous membrane with the hemorrhagic spots found on it, the congested state of the intestines, and the congested condition of the veins in general throughout the body—these morbid changes in the brain, and heart, and liver, and alimentary canal, and venous system of this unfortunate subject were all due to chronic alcoholism, that is, to chronic poisoning with alcoholic drinks.

But we should not forget that, in such cases as the foregoing and that which precedes it, a suddenly occurring paralysis of the vaso-motor nerves of the cerebral blood-vessels probably plays a very important part in the production of the cerebral hyperæmia that is found at the autopsy, and of the symptoms of the apoplectic fit itself. Trousseau says: "We know that . . . paralysis, or weakness of the vaso-motor nerves, causes relaxation of the coats of blood-vessels, blood-stasis, and consequently congestion." (Vide *Lectures on Clinical Medicine*, vol. i. p. 584, New Sydenham Soc. translation.) On this point consult also the last paragraphs in the last chapter on the influence of alcoholic intemperance, and the abuse of opium and other narcotics, as causes of apoplexy. The remarks which we have here made concerning the part played by vaso-motor paralysis of the cerebral blood-vessels in the production of apoplectic coma, in this case, are applicable to all the other cases related by us, in which the abuse of alcoholic drinks or of opium was a prominent cause of the apoplectic attack, and we make them here once for all. See also the comments on the preceding case, and the general summary of all these cases at the end of this chapter.

CASE IV.

Apoplexy both congestive and serous, (so-called,) the result of alcoholic intemperance and gluttony ; death ; autopsy.

Henry W., aged about 45, a New-York pilot, of very full and corpulent habit of body, reported to have been much addicted to the pleasures of the table and to excessive drinking, was seen to fall down suddenly upon the pavement, in a state of complete insensibility, while walking in the street, on the night of August 27th. A police-officer came immediately to his assistance and found him lying helpless, unconscious, and breathing stertorously, just where he had fallen. Additional help was at once procured in order to carry him to the station-house, but he died before getting there, although the distance was only a few blocks. It was supposed that he was recovering from a debauch when he was thus struck down with apoplexy. He did not have any convulsive movements.

Autopsy, by the author, twelve hours after death. The body was fat, muscular, and in good preservation, the shoulders broad, the chest large, the neck short and thick, the head large, the face, which was naturally full, swollen and livid, the conjunctiva injected, the pupils normal, the mouth drawn somewhat toward the right side.

Head.—The scalp bled freely while we were dissecting it off. There was a slight contusion on the back part of the head, received, probably, in falling to the pavement. While sawing off the skull-cap, considerable bloody fluid flowed out from the cranial cavity. On removing the dura mater, the visceral arachnoid membrane was seen to be raised up and to present a jelly-like appearance, especially at the vertex, in consequence of a very copious effusion of pale serum into the subarachnoid connective tissue, and into the sulci of the brain. The glandulæ Pacchioni were large and numerous The vessels of the pia mater were filled with blood to distention, or strongly congested. On slicing the brain, the substance was everywhere found to be strongly congested, that is, the bloody points on the cut surfaces were unusually large and numerous, furnishing blood enough to thoroughly besmear these cut surfaces. The consistence of the brain was natural, and its color good, except so far as it was affected by the blood which flowed out from the small vessels that were divided while slicing the organ. The ventricles were nearly empty. We found a small, globular, white tumor, having the size of a pea, and an apparently fibrous structure, projecting into the cavity of the right lateral ventricle, and attached by its side to the optic thalamus.

Thorax.—The lungs were entirely free from adhesions, contained more than the normal quantity of blood, and in their posterior parts exhibited strongly marked post-mortem congestion or suggillation, but presented no other abnormity worthy of remark. The heart was somewhat hypertrophied and its apex rounded off. Its mitral and aortic valves were slightly thickened. In other respects it was normal. The blood was fluid.

Abdomen.—The liver was congested, but otherwise sound. The stomach was moderately distended and exhibited a faint reddish tint externally. It contained, besides gases, between six and eight ounces of matter consisting of a brown-colored fluid, mixed with partially digested food, which exhaled the odor of the so-called sarsaparilla soda-water that is ordinarily sold in porter-houses. Its mucous membrane was reddened and softened throughout. It did not exhibit any thickening, but in spots seemed thinner than natural. The intestines were congested. The condition of the spleen and kidneys was not recorded.

Comments.—The subject of the foregoing history presented in his person a classical type of the so-called apoplectic configuration. He was thick-set, quite fleshy, very plethoric, large-headed, full-faced, and bull-necked. His friends were not surprised to learn that he had a stroke of apoplexy.

The question might be raised whether he was not in reality struck down by epilepsy instead of apoplexy. Indeed, this question presented itself to us when we made the post-mortem examination. But after careful inquiry we concluded that epilepsy formed no part of his disorder. He had not been subject to epileptiform attacks of any kind, he did not utter any cry when seized with the fatal fit, he did not froth at the mouth, he did not bite his tongue or lips, he did not pass his urine or fæces, and he did not have convulsions. On the contrary, he presented exactly the symptoms of a very severe attack of apoplexy; and there is no good ground for doubting that his disease really was apoplexy.

The etiology of the disease in this case was sufficiently obvious. The causes were two in number: 1. Habitual excess in eating; 2. habitual excess in drinking. The former induced a chronic state of overfulness and increased tension in all the blood-vessels of the body, including those of the brain, or a state of habitual plethora. The latter occasioned congestion and stagnation of blood in all the veins of the body, including those of the head, and atrophy of the brain with great disorder of its vaso-motory apparatus, which terminated in anæmia and sudden paralysis of the nerve-fibres and ganglion-cells of the organ, or apoplexy.

This case also presented the anatomical lesions which are supposed to characterize both the congestive and the serous forms of this disease. The face was purple from venous congestion. The sinuses of the dura mater, the vessels of the pia mater, and of the substance of the brain, were all distended with blood, or congested. Besides, there was well-marked œdema of the cerebral meninges. The subarachnoid connective tissue was distended with serum on the convex surface of the hemispheres, especially at the vertex, so that the arachnoid membrane presented a jelly-like appearance. It is probable that there was also œdema of the brain-substance, and that a sudden transudation of serum into the peri-vascular connective tissue of the brain-substance had compressed the capillaries and caused anæmia in them, while the larger vessels, and especially the cerebral veins, were still distended with blood. Thus we see how a state of sudden anæmia of the nerve-fibres and ganglion-cells of the brain is compatible with a state of hyperæmia of all the larger blood-vessels belonging to the brain and its membranes.

Case V.

Apoplexy both congestive and serous ; death ; autopsy.

Mary Mahr, aged about 56, was found lying dead on the floor of her room, on the afternoon of Nov. 9th. A short time previously, namely, at 12 o'clock, she had been seen by several of her neighbors, and appeared to be in good health. It was said by all who knew her that her habits were good and that she had always been healthy.

Autopsy, by the author, Nov. 10th. Cadaver rather fleshy and rigid. Pupils natural.

Head.—Brain and its membranes congested. There is an abundant quantity of serum beneath the visceral arachnoid membrane, especially at and about the vertex. The glandulæ Pacchioni are large and distinct. The arachnoid is opaque and a good deal thickened. The ventricles contain some serum. The cerebral convolutions and brain itself as a whole appear to be considerably shrunken; and its substance is rather soft.

Thorax.—Old pleuritic adhesions on both sides, but they are more extensive and more firm on the right than on the left side. There are some dark-colored tuberculous concretions in each lung, and old tuberculous cicatrices at apex of left lung. Both lungs are congested. Heart natural, except that the mitral and aortic valves are somewhat thickened. Foramen ovale completely closed.

Abdomen.—Liver congested and rather nutmeggy. Gall-bladder contains several small biliary calculi. Spleen normal. Kidneys congested. Stomach and intestines congested. The stomach contains six or eight ounces of food undergoing digestion. Its mucous membrane is slightly reddened throughout, and appears to be rather thick and soft. The congestion of the abdominal and thoracic organs above mentioned was principally venous.

Comments.—This case shows that temperate and discreet people are not entirely exempt from sudden death attended by apoplectiform phenomena. However, there can be no question but that they are much less likely to be attacked with apoplexy than those who indulge to excess in food and drink.

The post-mortem examination revealed the fact, that *senile atrophy of the brain* was present. Indeed that organ looked as if it belonged to a person of considerably greater age than the deceased was stated to be. It is not difficult to conceive that this softened and shrunken condition of the brain had materially assisted in producing the apoplectic stroke.

Again, the history of the last two cases clearly shows that the boundary between congestive and serous apoplexy is not sharply defined. In both of them alike cerebral congestion and subarachnoid effusion of serum were prominent post-mortem lesions. Hence, viewing these cases in the light that was shed upon them by post-mortem examination, we say that in each of them apoplexy was present in a serous as well as in a congestive form. Thus we perceive how closely these two varieties of apoplexy are allied to each other. It is totally impossible to distinguish them clinically; and when we consider them from a purely anatomical point of view, we find it equally difficult to separate them from each other. The so-called serous apoplexy is almost always accompanied by engorgement of the cerebral blood-vessels, although the so-called congestive apoplexy may, at least in typical cases, be accompanied by the effusion of but little serum beneath the visceral arachnoid, or in the ventricles, or in any other situation about the brain. The cases of serous apoplexy are, for the most part, also congestive in character, and therefore the serous should generally be looked upon as only a variety of the congestive form of the disorder.

Case VI.

Serous apoplexy occurring without warning, in an intemperate subject, while working at his trade; death in about fifteen minutes; autopsy.

Samuel B. W., aged 42, a tinsmith of intemperate habits, fell suddenly in the shop, as if he had been struck down by a blow, while standing at his bench at work, on the afternoon of July 7th. He was insensible, his breathing stertorous, and he died in about fifteen minutes. A woman who witnessed the occurrence told us that he had no convulsive movements. He had appeared to be in usual health all day, working and partaking of food without complaint. He was so much addicted to drink that it was said he spent most of his earnings in that way. A woman with whom he formerly boarded told us he had "fits" several times before. The shop in which he worked was not hot. It was a basement with windows both in front and rear, through which the air could circulate without hinderance. The temperature was not greater than usual on the day he died.

Autopsy, by the author, nineteen hours after death. Body rather fat and in a good state of preservation. The post-mortem rigidity was strong. The pupils were symmetrically dilated.

Head.—While taking off the skull-cap and removing the brain, between four and six ounces of serum stained with blood either flowed away or were collected in the occipital fossa. On turning back the dura mater, the visceral arachnoid was seen to be raised up, and to present a jelly-like appearance from an abundant effusion of pale serum into the subarachnoid connective tissue, which filled up the furrows of the brain and overflowed the convolutions. The glandulæ Pacchioni were more numerous and larger than natural. The arachnoid exhibited some pearly opacity on the convex surface of the hemispheres, and there the subarachnoid œdema was the most abundant. The ventricles contained three or four spoonfuls of pale serum. The vessels of the brain did not contain more than the normal quantity of blood, that is to say, they were not overloaded with blood. The substance of the brain, both cortical and medullary, was rather softer than natural. This softening was uniform in all parts of the organ, but did not exist in a high degree. The surface of a section was pale, moist, shining, and rather exsanguinated in appearance.

Thorax.—The lungs were free from adhesions. They were moderately œdematous, especially in their posterior or depending portions. On cutting into them some pale serum flowed away. In other respects the lungs were sound. The pericardium was adherent to the heart throughout its whole extent, whereby the pericardial surface was completely obliterated. It required considerable force to peel off the pericardium. The heart was considerably hypertrophied. There were some semi-cartilaginous deposits, the results of an old inflammation, in both segments of the mitral valve. They were most abundant near the free margin and produced considerable thickening. The semilunar valves of the aorta were also thickened by a similar process. Still, the amount of thickening was not so great as to produce insufficiency of either of these valves. The tricuspid and the valves of the pulmonary artery were natural. There were no heart-clots, and the blood was fluid in the great vessels.

Comments.—The suddenness of the attack, its freedom from shrieking, frothing at the mouth, convulsive movements, and other phenomena of epilepsy, the state of insensibility, the stertor, the rapidity with which death ensued, and the cerebral lesions revealed by the autopsy, show conclusively that this man's disease was apoplexy.

But in one respect, at least, this case differs considerably from those which we have previously related, namely, the brain was found to be free from congestion and rather exsanguinated at the time the autopsy was made, while in the other cases the vessels of the brain and its membranes were found to be engorged with blood. Besides, the œdema of the meninges was more strongly marked in this case than in the others. There was also œdema of the brain-substance, for the surface of a section was found to be pale, moist, shining, and rather exsanguinated in appearance. The morbid lesions presented by the brain in this case were atrophy, œdema, softening, and anæmia of its substance; and having them in view, it is not difficult to comprehend how the apoplectic stroke occurred. If the œdema of the brain-substance occurred suddenly, the cerebral capillaries were compressed with corresponding suddenness, the ganglion-cells and nerve-fibres deprived of blood, their functions arrested, and a state of apoplexy produced. In all probability that is precisely what did occur. It is possible, however, that a sudden vaso-motor spasm of the cerebral capillaries aided in producing the attack. On this hypothesis the sudden anæmia of the cerebral capillaries which occasioned the apoplectic symptoms was due partly to compression from œdema of the brain-substance, and partly to vaso-motor spasm of the cerebral blood-vessels.

When did the œdema of the brain and its membranes, which constituted so important a feature at the autopsy of this case, occur? Was this serum wholly effused before death? Or was part of it effused after death? Who can decide these questions? A positive answer to any of them is impossible in the present state of our knowledge. We think, however, that this œdema of the meninges and brain-substance occurred partly before and partly after death. We believe that, for some considerable time, it was preceded by chronic cerebral hyperæmia, for we know that habits of alcoholic intoxication, such as this man possessed, always induce a congested state of the cerebral blood-vessels. We do not think that this cerebral hyperæmia had yet disappeared when he was attacked with apoplexy. It is probable that the œdema began with sudden effusion of serum into the perivascular connective tissue of the brain-substance, and this occurrence produced the apoplectic phenomena in the way mentioned above. But the transudation of serum did not stop at this point. The watery or amorphous constituents of the blood continued to be exuded through the walls of the swollen blood-vessels into the subarachnoid connective tissue, into the ventricles, and into the substance of the brain, either from some functional disorder in the walls of these vessels, or from some change in the composition and dynamical state of the blood itself; and this process of serous transudation did not cease with death, but continued while the vascular contractility lasted. Thus it happened that the congested cerebral vessels became, as it were, depleted or partially emptied by giving up a portion of their contents, both before and after death, and thus they were found to present a non-turgid appearance at the autopsy. If the apoplectic attack was attended with vaso-motor spasm of the cerebral blood-vessels, it is easy to understand how the transudation of serum was produced and why it continued after death.*

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* In connection with the subject of cerebral œdema the following possesses considerable interest. *Marcé* has shown by experiment that the brain is capable of absorbing water to the amount of half the weight of the portion experimented with. He also has constantly found that in brains whose membranes were infiltrated with serum, the gray matter contained a larger proportion of water than in the normal state; for example, 85.9 per cent, instead of eighty per cent. (Vide *New Sydenham Soc. Year-Book*, 1860, *p.* 163.)

The post-mortem examination also revealed a peculiar condition of the lungs which may serve to throw some light upon the question as to the time when most of the cerebral œdema occurred. This peculiar state of the lungs was œdema of the posterior or depending portions of these organs instead of post-mortem congestion of the same parts, or, in other words, pulmonary œdema had taken the place of post-mortem congestion. This pulmonary œdema must, for the most part, have occurred after death, for the amount of it was so great as to be quite incompatible with life. If it occurred post-mortem, the presumption is that it occurred some time after death, namely, after the post-mortem congestion of the lungs had taken place which was represented at the autopsy by this transuded serum. We should here remark that there was no anasarca or general dropsy. Now, if the pulmonary œdema, for the most part, did not occur until after death, why may we not safely hold the opinion that the cerebral œdema, for the most part, did not present itself until the subject was in articulo mortis or had expired? We should also observe that the atrophy of the brain was well marked, for, otherwise, this excessive œdema of the membranes and substance of the organ could not have occurred.

Case VII.

Apoplexy, serous, so-called; caused by alcoholic intemperance; death in ten minutes; venesection was employed without any benefit; autopsy.

Amos F. P., aged 36, occupation unknown, while walking along the street on the afternoon of November 3d, fell down insensible and had stertorous breathing. A physician was immediately called, who bled him in the arm, but he died in about ten minutes. He did not have any convulsive movements. He was said to have been much addicted to the use of ardent spirits.

Autopsy, by the author, about twenty-four hours after death. The body was pale, emaciated, and not œdematous. The rigor mortis was strong.

Head.—The glandulæ Pacchioni were well developed. There was an abundant quantity of pale serous effusion beneath the visceral arachnoid, where it covers the convex surface of the hemispheres, which filled the furrows of the brain, overflowed the convolutions, and raised the arachnoid so as to give it a jelly-like appearance. This œdema was most abundant at the vertex. The arachnoid was also opaque and thickened by an old deposit of plastic exudation, for the space of about a square inch, on the convex surface of the right cerebral hemisphere. The vessels of the pia mater and substance of the brain contained more than the normal quantity of blood, that is, they were congested. The consistence of the brain-substance was rather firmer than it should have been, but its color was normal. The vessels of the brain-substance were all dilated. The ventricles contained about two dessert-spoonfuls of pale serum.

Thorax.—The lungs contained considerably more than the normal quantity of blood. In the right lung we also found a few miliary tubercles that were obsolete or in the course of repair. There was an abnormal layer or quantity of fat on the exterior of the heart, but in all other respects it was natural. The blood contained in the heart, venæ cavæ, and other large vessels was fluid.

Abdomen.—The liver was normal in size and shape, but on its surface, which was smooth, we found several yellowish spots of commencing fatty degeneration. These spots were of various sizes, and extended into the substance of the liver to various depths. The spleen had a normal size; but its investing peritoneum near one edge was much thickened and presented a yellowish-white color. The left kidney was rather larger than natural, and intensely congested. Its cortical substance or portion was rather thin, and there was an abundant quantity of adipose tissue in its pelvis. In a uriniferous tube we found a small calculus. The right kidney had a normal size, but it also was congested. In both kidneys the calyces were large, the apices of the cones appeared to be unusually distinct as they projected into the calyces. The pelvis and calyces of each kidney were full of urine bearing a considerable resemblance to water-gruel in appearance. There was no anasarca in any part of the body. The stomach contained about four ounces of food partially digested, and mixed with some liquid which exhaled the odor of rum. Its mucous membrane had a rose color in the fundus, but in the other parts of the organ it was pale. It was well coated over with viscid, glairy mucus. It was also thickened and softened throughout its whole extent, and resembled thin, pale, rotten leather. The bladder was about half full of urine, which did not exhibit any abnormal appearance to the eye. It was not subjected to chemical or microscopical examination. The kidneys were embedded in a good deal of adipose tissue, the mesentery also was decidedly fatty, while the body generally was thin, that is, the connective tissue in general contained but little fat. The voluntary muscles were rather paler than natural.

Comments.—This case belongs to the same category as the last. This subject was struck down by an attack of so-called serous and congestive apoplexy which quickly destroyed his life. There is no doubt that this attack was long preceded by a state of chronic hyperæmia with progressive atrophy of the brain, resulting from excessive indulgence in alcoholic drink, such as we have found in other topers when their bodies were examined after death. But the hyperæmia of the brain in this case had mostly disappeared when we made the autopsy; for the state of his blood and of the walls of the cerebral vessels was such that serum was readily effused in large quantity from the engorged blood-vessels into the subarachnoidean connective tissue, the ventricles and substance of the brain. The œdema of the brain-substance probably occurred at the same time as the apoplectic fit, and caused it by compressing the cerebral capillaries and thus inducing anæmia of the nerve-fibres and ganglion-cells of the organ. The effusion of serum, however, did not stop here. It, doubtless, continued through the article of death, and, after that, while the cooling of his body went on. But it did not continue long enough to bring the contents of the cerebral blood-vessels entirely down to the normal standard in respect to quantity; for these vessels were still found to be rather more distended with blood than they should have been at the autopsy. They also showed in their dilated appearance the evidence of the chronic congestion to which they had long been subjected.

This man's apoplexy was undoubtedly caused by drinking spirituous liquor. This evil habit induced a state of chronic hyperæmia with progressive atrophy of his brain, and finally, through intercurring œdema of its substance, produced anæmia of its capillaries and paralysis of all its functions. The kidneys were also found to be somewhat diseased at the autopsy. Now, it may be claimed that death in this case was mainly due to the disorder of these organs, and that it was produced by the poisoning of his blood with urea alone. It is doubtless true that urea is a poison which, by virtue of its own inherent properties, is capable of destroying life; but whenever it does so, at least so far as we have any positive knowledge, the symptoms of coma are gradually and not instantaneously produced. Urea is not one of the poisons which instantaneously destroy the consciousness, as we may readily see in any case of Bright's disease that is far advanced, and has much urea retained in the blood. And in these cases when death results from an uræmic intoxication, that is undoubted, the coma is always gradually developed. It is probably true that while the disorder of the kidneys which this man had was not the sole cause of his apoplexy, it assisted materially in causing this disease, by producing a tendency to the sudden occurrence of œdema in the perivascular connective tissue or spaces of the brain-substance and membranes, just as we frequently see œdema suddenly present itself in other parts of the body in cases of Bright's disease. It is probably true that, in this way, the disorder of the kidneys in this man's case coöperated with other causes in the production of the apoplectic fit. But this disorder of his kidneys was itself also a result of alcoholic intemperance.

The post-mortem examination of this subject presents several interesting features which are not directly connected with the cerebral lesions. They are, *firstly*, the commencing fatty degeneration of the liver; and *secondly*, the morbid state of the stomach, and especially of its mucous membrane. That membrane was in a state of chronic catarrhal inflammation, due to chronic irritation from alcoholic liquors, which was evinced by its being coated over with much tough, glairy mucus, by its being altered in color, and by its being thickened and softened. It was changed in structure so much that it bore considerable resemblance to pale, rotten leather. According to the author's experience, this is the typical form of the old toper's stomach, that is, the kind of stomach which is produced by chronic alcoholism. It is hardly necessary to say that such a state of the gastric mucous membrane cannot be produced by a single debauch. It requires for its development long-continued contact with diluted alcohol, or with some other liquid substance having irritating properties of a similar character.

Bloodletting was employed in this case without benefit. Perhaps it hastened the fatal result. Indeed, it is difficult to see what good bleeding can do in such cases as the foregoing, while it is easy to understand why the loss of blood may do such people much harm.

CASE VIII.

A middle-aged woman died suddenly of apoplexy, as it was supposed; her habits were intemperate; autopsy; the lesions pertaining to so-called serous and congestive apoplexy, and to chronic alcoholism, discovered.

Mrs. Mary B., aged about 35, married, reputed to have been a hard drinker for several years, was found about 7 o'clock P.M., Oct. 5th, sitting on a chair in her room quite dead, with her head leaning back against the wall. She was seen a short time previously in a grocery store purchasing milk for her husband's supper. She was said to have been drinking pretty freely during the afternoon before her death, and seemed to be in usual health. We could not learn that she had ever suffered from any severe sickness except the cholera in 1849. Her husband slapped her face a few days before death, and this produced a black eye, but no other inconvenience. Her dead body was discovered by a female acquaintance.

Autopsy, by the author, Oct. 6th, 12 o'clock M., about seventeen hours after death.—The body was spare, pale, and in good preservation. The rigor mortis was tolerably well marked. The right eye was blackened by ecchymosis, which appeared to be of several days' standing, and there was also a small bruise on the scalp at the back part of the head.

Head.—The sinuses of the dura mater, and the blood-vessels of the brain and its membranes generally, were distended with blood. The glandulæ Pacchioni were very large and distinct, especially at the summit of the left hemisphere, where they produced firm adhesions between the visceral arachnoid and the dura mater. On slicing, the medullary portion of the cerebrum was found to present a "sieve-like" appearance, from chronic dilatation of its blood-vessels. The cortical substance, particularly on the posterior lobes of both hemispheres, seemed to consist of two distinct layers, of which the outer was about half as thick as the inner. The substance of the whole brain was very firm. The subarachnoid effusion of serum was very abundant. On the convex surface of the hemisphere, it filled the sulci, giving the arachnoid a gelatinous look. The cerebrum had a firmer consistence than the cerebellum.

Thorax.—The lungs contained more than the normal quantity of blood, but in other respects were sound. The heart was natural. The condition of the blood was not recorded.

Abdomen.—The liver was large, pale, fatty, and flabby. The stomach contained several ounces of food undergoing digestion. The odor of spirits was not detected. The gastric mucous membrane was thickened and softened, pretty uniformly, throughout its whole extent. It was also colored pale-red in streaks or stripes, running lengthwise from the cardiac to the pyloric orifice. Except these streaks, its color was uniformly pale. Of the spleen, kidneys, and intestines no abnormity was recorded.

Comments.—Was the disease in this case apoplexy? We think it was. The stroke occurred while she was sitting in a chair. She leaned backward against the wall of her room near which the chair happened to stand, and this circumstance saved her from falling to the floor. The posture of her dead body, and the placid expression of her countenance, which was that of quiet slumber, show conclusively that coma was suddenly produced and that she did not have convulsions. It is obvious that convulsive movements would have thrown her body from a sitting posture on a chair down to the floor. She did not bite her tongue, nor froth at the mouth, nor pass her urine, nor present us with any of the symptoms of epilepsy. We therefore have good reason for believing that her disease was apoplexy. The cause was alcoholic intemperance. The cerebral lesions were chronic hyperæmia and dilatation of the blood-vessels of the brain and its membranes, œdema of at least the membranes, and atrophy with induration of the brain-substance. It is probable that anæmia of the cerebral capillaries and arrested function of the nerve-fibres and ganglion-cells were produced, in this case, in just the same way as in the other cases which we have related.

Case IX.

So-called serous and congestive apoplexy; death; autopsy; ossification of falx cerebri; copious effusion of serum beneath the visceral arachnoid, and in the ventricles; anatomical evidences of chronic congestion of the brain-substance; walls of the lateral ventricles softened.

William M., aged 58, by occupation a steward and bartender, was found lying on his right side in bed at the Northern Hotel, where he was employed, quite dead, about 8 o'clock P.M., August 9th. He was last seen alive on Sunday evening, Aug. 8th. He then appeared to be in good health. But he was a free liver. When found, the body was still warm.

Autopsy, by the author, Aug. 10th, at 12½ o'clock P.M.—Posture of body still undisturbed. It lies partly on right side and partly on back; legs (both) rigidly flexed; left arm lying straight by side; right arm bent; right thumb also bent beneath the forefinger; eyelids closed, pupils somewhat dilated, (symmetrically;) countenance not distorted; bed-clothing not disturbed, as they probably would have been if convulsions had occurred.

Head.—There is a lamina of osseous matter deposited on the falx cerebri. It is three fourths of an inch in length and half an inch in breadth. On the convex surface of both cerebral hemispheres an abundant quantity of pale serum has been effused beneath the arachnoid membrane. This serum fills the sulci, overflows the convolutions, and raises up the arachnoid, so as to cause it to present a gelatinous appearance. The veins of the pia mater are considerably distended or congested, and the smaller twigs are beautifully injected with blood. The glandulæ Pacchioni are very distinct. The arachnoid exhibits more or less pearly opacity in several places. Each lateral ventricle contains rather more than half an ounce of pale serum. The plexus choroideus on each side presents a beaded appearance, from effusion of limpid serum into its connective tissue, (vesicles.) On slicing down the brain, the punctiform spots (puncta vasculosa) are seen to be more numerous and distinct than usual, and the surface of a section presents a somewhat "sieve-like" appearance, when freed from blood. There is also an abundant quantity of serum about the medulla oblongata and cerebellum. The substance of the cerebellum presents morbid appearances analogous to those of the cerebrum. The brain-substance was not softened anywhere except in the walls of the lateral ventricles. There the brain-tissue was somewhat pulpified and white in color. No pus was present.

Thorax.—No pleuritic adhesions. Lungs (both) contain pigmentum nigrum in excess, and a few old miliary tubercles in a calcareous state.

Right lung congested, principally *post-mortem*, however, since the body was found lying partly on the right side. Right pleural cavity contains six or eight ounces of bloody serous effusion, (also *post-mortem.*) Heart exhibits rather more fat on its exterior than usual; and is somewhat hypertrophied. The mitral, aortic, and tricuspid valves are somewhat thickened. The foramen ovale is completely closed. The right chambers of the heart, and the large vessels connected therewith, were distended with blood.

Abdomen.—Liver contains more than the normal quantity of blood. Spleen natural. Stomach contains a few ounces of dark-colored, oily liquid, having a sour, pungent odor. Its mucous membrane is more or less reddened, thickened, and somewhat softened throughout. Kidneys congested, (venous.) Intestines congested, (venous.)

Comments.—There was no suspicion that this man committed suicide, or that he was the subject of foul play. We have no reason for attributing his death to any other than natural causes. His case, doubtless, belongs to the same category as those we have already related.

The cause of the disease was, doubtless, high living, together with the abuse of spirituous liquors. The cadaver had the appearance of belonging to a hard drinker. Still the deceased was not considered to be intemperate by his acquaintances. He probably ate heartily, and drank freely and steadily.

The anatomical lesions revealed by the autopsy in this case agree, in all essential respects, with those which were found in our other cases of apoplexy. There was chronic hyperæmia of the venous system of the whole body, but especially of the brain and its membranes. The brain itself was atrophied, the vessels of its substance dilated by this congestion, and there was copious effusion of serum in the subarachnoid connective tissue, in the ventricles, and probably in the perivascular spaces of the brain-substance also. The chronic cerebral hyperæmia finally produced œdema of the brain-substance, compression and corresponding anæmia of its capillaries, and arrest of function in its nerve-fibres and ganglion-cells, or, in other words, a state of apoplexy. But the medulla oblongata also became paralyzed in the same way, the respiratory movements immediately ceased, and death ensued.

Case X.

Chronic alcoholism ; sudden death ; autopsy.

An unknown colored man, aged about 30, was brought to the Essex Market Prison by the police, on the morning of November 12th, in a state of stupor occasioned by drunkenness, as it was supposed. He was committed on the charge of vagrancy and intoxication, and placed in a cell to sleep off the effects of his debauch. Some hours afterward, he was found to be dead. When committed he could still be roused from the stupor.

Autopsy, by the author, a few hours after death.—The external examination showed that he had the clap, and a chronic ulcer on his left leg, of large size.

Head.—The scalp was congested. The dura mater was thicker than natural, the result of an old morbid process. The veins of the pia mater were turgid with blood, and their little twigs were minutely injected. Beneath the visceral arachnoid, especially about the vertex, we found an abundant quantity of pale serum, which filled the sulci and raised the arachnoid, giving it a pale, jelly-like appearance. There was a little pale serum in the ventricles, and an abundant quantity of similar fluid about the cerebellum. The substance of the brain contained more than the normal quantity of blood, and the little vessels traversing it appeared to be larger than natural, or dilated. Its consistence was very firm. Externally, the cerebellum was rather paler than the cerebrum, but its consistence was equally firm. About six ounces of serum and blood either flowed out from the cavity of the skull, or collected in the occipital fossa, during the examination of the brain.

Thorax.—The right lung was adherent to the chest, throughout its whole extent, by old, grayish-white, cellular false membrane. The left lung was entirely free from adhesions. Both lungs contained more than the normal quantity of blood, and their posterior or depending portions were also œdematous. The heart was a little larger than natural, but presented no other abnormity.

Abdomen.—The liver was enlarged to nearly twice the normal size, its color was darker than natural, its texture somewhat softened, and it contained more than the normal quantity of blood. It exhibited a coarsely granular fracture, but its surface was smooth. The spleen was also enlarged, dark-colored, and somewhat softened. The kidneys were larger than natural, pale, and mottled. On laying them open with a scalpel, the cortical substance was found to have a yellowish color, and its cut surface was dotted over with a great many little pale-red spots. The pyramids were congested. The stomach was larger than natural. Its mucous membrane was reddened, thickened, and softened. The odor of spirits was not present. The intestines were congested with venous blood. The voluntary muscles did not exhibit any abnormity.

Comments.—It may be that the police were mistaken in regard to the cause of this man's stupor. It may have been produced, not by drunkenness, but by anæmia of the nerve-fibres and ganglion-cells of his brain, resulting either from venous hyperæmia and stagnation of blood in the cerebral capillaries, or from œdema of the perivascular connective tissue of the brain-substance and consequent compression of its capillaries; or his stupor may have been produced by blood-poisoning from urea, as his kidneys were both fatty and congested; or all these causes may have coöperated in producing the stupor, coma, carus, and death. We have no doubt that this man died, not from acute poisoning by alcohol, but from disease operating in the way mentioned above. We may here remark that the police of New-York not unfrequently make a sad mistake in taking it for granted that a person whom they chance to find in the street in a stupefied condition, is drunk, and consigning him to the cells of a prison instead of the wards of a hospital. Sometimes, in this way, no doubt, they have allowed people to die uncared for in the solitude of a prison-cell, whose lives might have been saved.

This was not a case of apoplexy. The coma was not produced in that sudden manner which characterizes an apoplectic fit. Still it belongs to the same category as that disease. The causes and post-mortem lesions were substantially the same as those observed in many of the cases which we have related, and the phenomena also were similar; the only difference being, that in the one the coma was gradually, and in the others it was suddenly produced. No doubt coma would have been suddenly developed in this case also, if an anæmic state of the nerve-fibres and ganglion-cells of his brain had been suddenly produced; and death was occasioned in substantially the same way as it is in cases of apoplexy. First, the functions of the cerebrum were suspended, one after another, until this part of the brain became completely paralyzed; then the medulla oblongata was invaded, its functions suspended, the respiratory movements arrested, and death produced.

The cause of this man's sickness and untimely death was alcoholic intemperance. The post-mortem lesions bear a strong resemblance to those which we have found in cases of an analogous character. There was a state of chronic venous congestion in all the body, marked hyperæmia of the brain and its membranes, with chronic dilatation of its blood-vessels, œdema of the meninges, the ventricles, and probably of the brain-substance also, atrophy of the brain itself, with induration of its substance, and fatty degeneration of the kidneys. The renal lesion, in all probability, induced a poisoned state of the blood from retained urea, and the poisoned blood aided in producing lethargy, coma, carus, and death.

CASE XI.

Chronic alcoholism; sudden death from the occurrence of congestive and serous apoplexy; autopsy.

Henry H., aged 43, a tailor, had been a hard drinker for many years.

On the afternoon of Sept. 3d he was turned out of a "grocery" in the neighborhood of where he lived, because he was drunk. On his way home, which was a distance of but a few doors, he fell down upon the sidewalk. He managed to get up, and finally to reach his boarding-house unaided, but there he fell again in the hall. In a few minutes after the last fall, he died.

Autopsy, by the author, about twenty-two hours after death. —The body was thin, *i. e.*, rather emaciated, pale, and in good preservation. The post-mortem rigidity was entirely absent. The right pupil was dilated, and the left natural.

Head.—The skull was very thick. The glandulæ Pacchioni were unusually large, numerous, and distinct, and formed some pretty strong adhesions between the dura mater and visceral arachnoid. Over the whole convex surface of both hemispheres, pale serum had been effused beneath the arachnoid, so as to raise it up, and give it a gelatinous appearance. The quantity of serous effusion was more abundant at the vertex than elsewhere. It occupied the subarachnoid connective tissue, filling up the sulci, and covering the surface of the convolutions. The ventricles contained a little serum stained with blood. The vessels of the pia mater contained more than the normal quantity of blood. The vessels of the substance of the brain also contained more than the normal quantity of blood. The consistence of the brain was very firm. On carefully slicing it, the thin sections of the white substance were found to present a "cribriform" or sieve-like appearance, from chronic enlargement of the little vessels which supply the interior of the brain with blood.

The thorax and abdomen were not opened, because the head was examined only for the purpose of ascertaining whether the skull was fractured, and whether blood had been extravasated within the cranium.

Comments.—The injury which this man received in falling, in the shape of concussion or commotion of his brain, doubtless assisted in producing the fatal coma. It probably determined the occurrence of œdema of the brain-substance, with consequent compression of its capillaries, and anæmia of its nerve-fibres and ganglion-cells.

At the autopsy the pupil of the right eye was found to be dilated, while that of the left eye was natural. No reason for this want of symmetry was discovered.

The substance of the brain in this case, as in several of the preceding ones, was found to be firmer than natural, or indurated. Now, this hardened condition of the brain is met with so frequently in the bodies of spirit-drinkers, that there seems to be something more than an accidental relationship between them. Indeed, it is well known that diluted alcohol, when taken into the stomach, is absorbed therefrom unchanged; that commingled with the circulating blood it is distributed throughout the body, in the same unchanged condition; and that some considerable time is required for its conversion or elimination to take place. Furthermore, Percy's experiments show that alcohol manifests a much stronger affinity for the substance of the brain than for any other structure of the whole body in living animals. May not the cerebral induration, which is so often found at the necroscopy of drunkards, be due to the direct action of alcohol on the brain-substance?

In some of the cases which we have related, a little cerebral softening was found at the autopsy; it appears, however, to have been occasioned, not by an inflammatory process, but by the imbibition of serum in some instances, and by impaired nutrition in others.

CASE XII.

Sudden death from apoplexy, as it was supposed; general health good; habits not bad; autopsy.

John G. R., aged 33, a merchant in good standing, was found lying dead in his bed, at the Frankfort House, where he lived, on the morning of April 30th. He was said to have been addicted to drinking lager-beer, but was not a drunkard. His clerk testified that on the preceding evening he seemed to be in liquor. He went to bed between eleven and twelve o'clock. When found he lay on his right side as if asleep. The door of his room was locked on the inside; his money, watch, and clothes were undisturbed; and there was no evidence, nor even a well-grounded suspicion, that he had committed self-destruction, as his affairs were prosperous and health good.

Autopsy, by the author, April 30th, at two o'clock P.M.—Body that of a hearty man; muscular and adipose tissues well developed; rigor mortis well marked; a little bloody fluid flowing out of the left nostril, and some darkish-colored frothy fluid from the mouth.

Head.—The arachnoid exhibited pearly opacity, and also some thickening in spots, especially about the vertex. The veins of the pia mater contained rather more than the normal quantity of blood. The substance of the brain was natural in color and consistence. There was no unusual effusion of serum beneath the arachnoid or in the ventricles.

Thorax.—The right lung was large in volume, increased in weight, darkened in color, and an abundant quantity of blood mixed with air-bubbles flowed out on cutting into it. The darkening in color and increase in blood was greatest in the depending portion. The left lung was also large in volume, increased in weight and darkened in color, but not to the same extent as the right lung. It will be remembered that the subject was found lying on his right side, and it is therefore probable that the blood had settled to a considerable extent from the left to the right lung, subsequent to death, in consequence of gravitation. Some small spots of blood extravasated beneath the pleura pulmonalis were found at the base of the left lung; and on cutting into any part of this organ an abundant quantity of serum flowed away, that is, there was œdema of this lung. In other respects the lungs were natural. The heart was normal, except the foramen ovale, which was not completely closed; but the opening was too small to allow any considerable commingling of the blood in the auricles. There were no heart-clots, and the blood generally was fluid.

Abdomen.—The liver was hypertrophied, and congested with venous blood, but we did not discover any other abnormity. The stomach was large in size and distended with gas. It contained some liquid having a dark color, resembling beer, and without alcoholic odor. Its mucous membrane was much thickened, more or less reddened, and mammillated throughout its whole extent. The spleen, kidneys, and intestines exhibited venous congestion, but otherwise were sound.

Comments.—The mechanism by which death was produced in this man's case is, to say the least, obscure. On the day preceding death he was in good health, and, indeed, his health, generally speaking, had been always good. The principal lesions revealed by the autopsy were a state of general venous congestion of all the organs of the body, moderate hyperæmia of the brain and its membranes, and strong hyperæmia of the lungs. Now, what caused this intense venous congestion of the lungs? The flow of blood into the left side of the heart was not impeded by any disease of the pulmonary veins or of the heart itself. He was not strangled nor suffocated. We must, therefore, seek for the cause of this pulmonary congestion in some spontaneous disorder of the muscular apparatus by which air is drawn into and expelled from the lungs. This apparatus had, probably, ceased to act, or at least had performed its office but imperfectly for some time before his heart ceased to beat. The consequence was that the blood accumulated in the pulmonary artery and its branches so as to constitute pulmonary congestion; for it is well known that the blood-corpuscles cannot pass through the pulmonary capillaries into the pulmonary veins and left side of the heart until their carbonic acid has been given up and fresh oxygen has been imparted to them. Unless this change takes place, the venous blood stagnates in the lungs, and continues to accumulate there as long as the heart continues to beat. Now, the movements of respiration ceased to be performed in this case because the nerves of respiration were paralyzed, and they were paralyzed because the functions of the medulla oblongata, the nervous centre which presides over the respiratory movements, were arrested, together with all the other functions of the brain. Thus we find, by an analytic examination of the post-mortem appearances presented by this case, that, in all probability, the disease was apoplexy. It certainly was not epilepsy, for the undisturbed condition of the bed-clothing proved that there had been no convulsive movements of a general character. Moreover, his tongue was not bitten, and his urine was not passed during the attack.

The cause of his decease was doubtless a state of general plethora, which resulted from the free use of food, and the free consumption of beer. More congestion of the brain and its membranes would have been found at the autopsy if the substance of the organ had been sufficiently atrophied to permit the blood-vessels of the brain and its membranes to undergo dilatation. His brain was that of a comparatively temperate person. It exhibited but little if any atrophy, and in this respect presented a strong contrast to the condition of brain which was found in many of the other cases. The apoplectic phenomena were probably produced in the following way: There was strong venous congestion of the brain with stagnation of blood in the cerebral capillaries, the nerve-fibres and ganglion-cells failed to receive a sufficient supply of oxygenated blood to maintain them in a state of activity, the functions of the cerebrum were thus suspended, and finally those of the medulla oblongata, when death ensued.

Case XIII.

Sudden insensibility attended by convulsive movements; death from coma a few hours afterward; autopsy.

Michael W., aged 43, a poor laboring man, but with the habit of hard drinking, of long standing, was found in the street, on the 18th of June, in a state of unconsciousness from which he could not be roused, by a police officer, who had him carried to the station-house. A physician was called to him, who states that he was totally insensible and had convulsive movements, epileptic in character, which continued till death occurred, a few hours afterward. We could learn nothing more of his previous history.

Autopsy, by the author, June 19th, about twenty-four hours after death.—The body was pale and emaciated, and there was but little post-mortem rigidity.

Head.—The dura mater was more strongly adherent to the skull than usual, especially at the vertex. The sinuses of the dura mater, together with the veins of the pia mater, were found to be distended with blood. There was also a moderate quantity of pale serum in the connective tissue beneath the visceral arachnoid. The ventricles contained two or three spoonfuls of pale serum. The brain had a good consistence throughout. On carefully slicing it, the puncta vasculosa were found to be more numerous and larger than usual, and the small blood-vessels permeating the brain-substance were seen to be somewhat enlarged or dilated, apparently from chronic congestion. In other respects the brain seemed sound.

Thorax.—The pulmonary pleura on the right side was bound to the chest, throughout the greater part of its extent, by firm old adhesions. The left lung also was adherent posteriorly. Both lungs were intensely congested. In some of the lobules, or series of lobules, the congestion was so great that they contained but little air, and presented, on cutting into them, a dark-brown color with a tolerably well-defined margin, which vividly reminded us of pulmonary extravasation; but we did not find any coagula nor any laceration of the pulmonary texture. The lesion was due to very large accumulation of blood in some of the branches of the pulmonary artery, without there being any rupture of their walls. In other respects the lungs were sound. The mitral valve of the heart was somewhat thickened; not enough, however, to produce mischief, but in other respects the organ was natural. The blood was fluid in the heart and great vessels.

Abdomen.—The liver was large, fatty, and granular, but presented no other abnormity. The spleen was somewhat larger than natural; its peritoneal coat was slightly thickened, and had a bluish-gray color. The lining membrane of the stomach was coated over with an abundant quantity of mucus, mammillated, and slightly softened. No abnormity of the kidneys was noted. The voluntary muscles were paler and softer than natural, and flabby.

Comments.—The disease in this case was not apoplexy, but epilepsy or epileptiform convulsions; and we have introduced the account of it here for the purpose of contrasting its phenomena with those of apoplexy. The main difference between the symptoms of epilepsy and those of apoplexy is, that in the first-named disease general convulsive movements are present, while in the latter they do not occur; but in both alike the victim suddenly falls down insensible, and passes into a state of coma.

The subject exhibited many of the lesions which characterize chronic alcoholism. He had the toper's stomach, together with the fatty liver, and his voluntary muscles were likewise undergoing fatty degeneration. He also had chronic hyperæmia of the brain, and there was considerable atrophy of the brain-substance; for otherwise this hyperæmia of the brain and its membranes with chronic dilatation of its blood-vessels could not have been accompanied by as much œdema of the meninges and dropsy of the ventricles as were found in this case.

The pulmonary congestion which the autopsy revealed was no doubt largely due to the disturbance of the respiratory movements by the convulsions, and the non-aeration of the blood in the lungs which resulted from it. This is one of the ways in which epilepsy destroys life in those cases where death occurs during the fit. Indeed, the risk of death taking place from apnœa is considerable in those cases of epilepsy where the convulsions have a tetanic character.

Case XIV.

Sudden death from eclampsia occasioned probably by uræmia; autopsy; Bright's disease of kidneys, etc.

Maria J., aged 25, an Indian woman of abandoned character, and addicted to the excessive use of spirituous drinks, died suddenly on the evening of October 30th. On Friday, the 29th, she was badly beaten by a colored man with whom she cohabited, and fled for shelter to the abode of an acquaintance, where she got along very well until the next morning (Saturday) about ten o'clock, when she became delirious, and continued to be out of her head through the day. In the evening she was suddenly seized with convulsive movements, became insensible, and died in about one quarter of an hour, according to the testimony of the witnesses.

Autopsy, by the author, about ninety hours after death.—The body was stout, plump, not dropsical, and beginning to exhale the odor of decomposition. There was no post-mortem rigidity. The right eye and cheek were blackened, apparently from a recent bruise. The skin was abraded on the bridge of her nose, which was covered with a bit of adhesive plaster. There were cicatrices of old buboes in both groins. Her chemise was stained with blood which probably had been discharged from the vagina, and was menstrual in character.

Head.—The skull was thick. The vessels of the pia mater were turgid with blood, and the substance of the brain also contained more than the normal quantity of blood. The color of the brain-substance was natural throughout, and its consistence remarkably firm. There was no accumulation of serum in the ventricles or beneath the arachnoid.

Thorax.—The left lung was adherent to the chest at its apex, and the right lung also was fastened to the chest by some old false membrane. The bronchial mucous membrane was reddened, thickened, and coated over with dark-colored bloody mucus. The lower portion of the upper lobe of the left lung was intensely congested, that is, it had a dark, or reddish-brown color; it contained scarcely any air, it had a fleshy feel, an abundant quantity of serum flowed away on cutting into it, but it was not softened. The part of the left lung which presented this appearance was bounded by a well-defined margin, and extended from the root through to the periphery or pleura pulmonalis, and a subdivision of the left bronchus traversed its centre. When viewed externally, that is, through the pleura pulmonalis, its surface was seen to present a brownish color, and to be slightly depressed below the surrounding surface, closely resembling in both of these respects the atelectasis of infants. Besides this large piece of the left lung, there were isolated lobules in the same lung, and also in the right one, which exhibited precisely the same morbid appearances. The bronchial tubes were all pervious, that is, not plugged or stopped up. The heart was large, flabby, and had something more than the normal quantity of adipose matter deposited on its exterior. There was a large milk-spot or insula on the right ventricle. There were also white or pale-yellow coagula (polypi) in all the chambers of the organ. The valves were natural, and its muscular substance sound. The lungs exhibited no abnormity besides those mentioned above.

Abdomen.—The adipose tissue was superabundant in the walls of the thorax and abdomen. There was also more than the normal quantity of fat in the omentum and mesentery. The liver was considerably increased in size, and its edges were well rounded off. Its surface was smooth, its color generally paler, and its consistence firmer than natural. On its surface, particularly the convex, there were some yellowish-white or pale-yellow spots of various sizes and shapes, which bore a strong resemblance in looks to the fat of bacon or lard. All of these pale-yellow spots extended into the substance of the liver to various depths, and had a firm consistence; they were not due to the ordinary fatty degeneration of this organ. The gall-bladder contained several small calculi, of which the greater number were found in the anterior or large extremity, and the rest in the small extremity of it.

The spleen presented no abnormity worth mentioning. The stomach was large in size, and contained about a half-pint of whitish milk-like fluid. Its mucous membrane appeared to be normal. The contents of the intestines were generally thinner or more fluid than they should have been.

The kidneys were larger and paler than natural. Their capsules presented a parchment-like appearance in spots, and were peeled off more easily than natural. Their consistence was good, and their surface smooth. On laying the cortical substance open with a scalpel, the cut surface was seen to present a pale-yellowish or lardaceous color, pretty thickly dotted over with very small and irregularly-shaped bright-red spots. The pyramids presented no abnormity. We found some offensive-smelling urine in the pelvis of the right kidney, which bore a strong resemblance in color and consistence to water-gruel.

There were some old adhesions about the uterus, ovaries, and oviducts, the vestiges of old inflammatory action. One oviduct was found to be closed at both extremities, and serous fluid had collected in its cavity until a good deal of enlargement was produced, that is, dropsy of the Fallopian tube had taken place. There were some small fibrous tumors in the fundus uteri. The voluntary muscles appeared to be normal in size, color, and consistence.

Comments.—This woman had probably suffered severely from syphilis, as she had led an abandoned life, and had the scars of old buboes in both of her groins; and the lardaceous or waxy degeneration of her kidneys and liver, which was so strongly marked in the first-named organs, was doubtless due to the continuance of that disease in a constitutional form. The affection of which she died had its origin in syphilis; for it had induced a peculiar change in the structure of the kidneys, which constitutes an important variety of Bright's disease, and fatally impairs the functions of these organs. In consequence of this degeneration, the kidneys became unable to perform their office of separating urea from the blood, and a state of uræmic intoxication, or blood-poisoning from urea, occurred, which in turn produced general convulsions, and arrest of the respiratory movements from convulsive closure of the glottis together with convulsive contractions of the respiratory muscles in general, or death from apnœa. The history of this case has been related in this place mainly for the purpose of contrasting the phenomena and the post-mortem lesions which result from uræmic intoxication with those which result from apoplexy.

This woman's brain was not atrophied, its blood-vessels were not dilated, and its meninges were not œdematous. It is also probable that the congestion of the brain and its membranes was due, in part at least, to the convulsions. But her kidneys were far advanced in the lardaceous or waxy degeneration, and during life she had general convulsions of so severe a character that they proved fatal in about a quarter of an hour. The condition of her lungs denoted that death had suddenly occurred from apnœa, that is to say, the convulsions had suddenly arrested the movements and the function of respiration. She also had bronchial catarrh of at least several days' standing, which was denoted by the reddening and thickening of the bronchial lining membrane, and by its being coated over with an abundant quantity of mucus; and this bronchitis had doubtless assisted in producing the peculiar condition of the lungs which was found at the autopsy.

The next case also is an example of uræmic convulsions resulting from Bright's disease of the kidneys.

CASE XV.

Chronic disease of the kidneys without anasarca; uræmic convulsions; death; autopsy; albumen found in the post-mortem urine, etc.

Dudley Robbins, aged 21, born in the State of Maine, by occupation a sailor, was admitted to Bellevue Hospital Dec. 21st, in a state of coma with general convulsions occurring in paroxysms. His pupils were dilated and rather sluggish to the irritation of light. The convulsions continued, he grew gradually weaker, the coma became more and more profound, and he died Dec. 22d, at 11 o'clock A.M. The house-physician learned from a doctor who accompanied him to the hospital, that the deceased had been ill but a short time; that when he first saw him he was in convulsions and comatose, and that he advised his removal to the hospital.

Autopsy, by the author, Dec. 25, seventy-four hours after death. —Body in a good state of preservation, adipose tissue scanty, muscles well developed.

Head.—Glandulæ Pacchioni numerous, large, and distinct. Brain and its membranes congested. A moderate quantity of pale serum beneath the visceral arachnoid membrane, and filling the sulci over the whole convex surface of the cerebral hemispheres. Color and consistence of the brain good in every part of it. Ventricles nearly empty.

Thorax.—Strong old pleuritic adhesions on both sides of the chest, but they were most abundant on the right side. Lungs exhibited post-mortem congestion in their depending portions, and a single, small obsolete tubercle in the apex of the left one; but in other respects they were healthy. Heart natural; foramen ovale closed.

Abdomen.—Liver and spleen normal. Intestines decidedly empty of fæces, but in all other respects normal. Stomach contained several ounces of dark-colored fluid. Its mucous membrane had a pale color. The pyloric portion was mammillated, thickened, and softened. The urinary bladder was distended with urine, which had a yellowish milky color and exhaled a strong ammoniacal smell. On testing it with heat, I found that the milkiness gradually disappeared till toward the boiling point, when it became quite transparent and did not contain any flocculi; that by boiling it a dense cloud formed, which separated into flocculi of considerable size after continuing the boiling a little while; and that the flocculi were not redissolved by nitric acid. I therefore concluded that this post-mortem urine contained albumen in considerable quantity. Prof. Clark repeated this examination of the urine with substantially the same results. The kidneys were rather large in size, and flabby. Portions of their cortical substance had a rather paler color than natural; and some lobules had a darker color than others, due, perhaps, to post-mortem congestion. Their calyces and pelves were somewhat dilated. Their capsules peeled off more readily than natural. Some of the tubuli uriniferi were dilated, which caused the organ to look, when cut into, as if it contained sinuses.

The body was not dropsical. Neither the hands, feet, nor face were swelled, and there was no accumulation of serum in the pleural, pericardical, or peritoneal cavities. Larynx rather large in size; but there was no abnormity worth mentioning in the fauces, pharynx, larynx, and trachea. The coats of the urinary bladder were natural in color, thickness, and consistence.

Comments.—It should be observed that in nearly all the author's post-mortem examinations related in these pages the condition of the fauces, larynx, and trachea was carefully inquired into, although special mention of these organs is made in the last case alone. This special mention was generally omitted because these organs were found to be unaffected. If they had been found disordered in any way, it would have been described in connection with the case to which it happened to belong. The author makes the above remark once for all.

It is the fashion nowadays to make much of Bright's disease, and probably sometimes, perhaps oftentimes, the sudden occurrence of coma has been attributed to that disease when it ought not to have been; but in the case which we have just related there is no reasonable doubt but that the renal disorder was the cause of the convulsions, coma, and death. The kidneys were the only organs which presented lesions at the autopsy that would satisfactorily account for the convulsions, the coma, the carus, and the fatal issue. They were large, pale, flabby, and their capsules peeled off more readily than usual. The cortical substance was pale, and some of the tubuli uriniferi were dilated from obstruction so much as to form what looked like sinuses. The well-marked albuminous state of the post-mortem urine conclusively showed that the degeneration of the secreting portion of the kidneys had progressed so far that it no longer performed its accustomed office. Of the effete material or substances floating in the blood, urea was no longer duly secreted or separated from it, and from this retention or accumulation of urea in the system there resulted a state of blood-poisoning from urea which was attended by the phenomena above mentioned. We may also remark, in this place, that whenever Bright's disease suddenly produces loss of consciousness or coma, it does so either by inducing epileptiform convulsions, or rapidly occurring œdema of the brain-substance.

The other organs of this young man's body were remarkably free from abnormal appearances. The brain was not atrophied, and the moderate degree of cerebral hyperæmia which was present may readily have been occasioned by the convulsive movements. The heart was perfectly natural. The lungs were so free from congestion as to show that the convulsions had not interfered much with the respiratory movements, and that death had not been produced by apnœa. He had probably died because he was worn out by the convulsions, and because he could no longer take the amount of nourishment into his stomach which is necessary to sustain life.

We shall refer to this case again when we come to speak on the diagnosis of apoplexy.

CASE XVI.

Sudden death from softening of the brain, attended by coma and convulsions; autopsy.

Mr. Moran, aged about 50, was brought to Bellevue Hospital, Nov. 14th, in a state of unconsciousness, and laboring under convulsive movements. Nothing of his previous history was known at the hospital. He died in the evening, comatose and in convulsions.

Autopsy, by the author, sixty-five hours after death.—Body in good preservation; post-mortem rigidity absent; pupils dilated, but the right more so than the left.

Head.—Brain and its membranes congested. An abundant quantity of serum beneath the arachnoid gives that membrane a gelatinous appearance. On slicing the brain, its substance exhibits the "cribriform" appearance in a marked degree, from dilatation of the blood-vessels. The brain-substance also is everywhere decidedly softer than natural, but the softening is most marked in the corpus callosum, optic thalami, and corpora striata. A moderate quantity of serum in the ventricles. The softened brain-tissue has a white color. There is an excess of serum about the cerebellum, and the arachnoid membrane investing it is opaque and thickened.

Thorax.—Lungs congested. Heart adipose on its exterior and flabby. Foramen ovale closed.

Abdomen.—Liver fatty in spots. Spleen natural. Kidneys large, and their cortical portion is paler than natural. Stomach and intestines congested with venous blood. Mucous membrane of stomach mammillated and soft.

Comments.—When softening of the brain produces epileptiform convulsions or eclampsia, it destroys the consciousness and paralyzes the functions of the cerebrum as suddenly and completely as apoplexy. But when it destroys the consciousness without producing epileptiform convulsions, the patient sinks into coma not suddenly but gradually. If in such a case the coma occurs suddenly, without convulsions, the attack is apoplectic in character. Hence it is sometimes said, in speaking of softening of the brain, that it predisposes to the occurrence of apoplexy.

Case XVII.

Hypertrophy of the heart; sudden death, occasioned probably by apoplexy; autopsy.

Adolph M., a colored man, somewhat past the middle age, and of good habits, went to bed on the night of April 7th in apparently good health. As he did not come down to breakfast next morning, his room was entered, and he was found lying dead in bed. From the undisturbed condition of the bed-clothes, together with the posture and appearance of his body, we inferred that he had died without convulsions, and without a struggle. Nothing was found which would warrant a suspicion that he died from any other than natural causes.

Autopsy, by the author, at four o'clock P.M., April 8th, the day after death.—Body large, muscular, and well formed. Pupils widely dilated. Mouth drawn toward the right side, and this side of the face presented a puckered or contracted appearance as it does in cases of left-sided hemiplegia. Rigor mortis very strong.

Head.—The skull was very thick, and partially eburnized. The arachnoid membrane exhibited some pearly opacity. The substance of the brain was natural in color, consistence, and in the quantity of blood which it contained. It did not exhibit any serous or sanguineous effusion. There was, however, a moderate amount of venous congestion and œdema of the meninges.

Thorax.—The cartilages of the lower ribs were ossified. Both lungs were much congested and œdematous. Some old pleuritic adhesions were found behind and near the base of the right lung. The heart was very much enlarged, being nearly or quite six inches in its long diameter. Externally it presented some inflammatory milk-spots to view. The cavity of the left ventricle was much dilated, and its walls considerably thickened. The right ventricle also was dilated, but its walls were not much thickened. The auricles exhibited dilatation. The aortic valves exhibited a puckered appearance and were insufficient. The mitral valve was a little thickened. The other valves were healthy. The right side of the heart was full of blood. There were no heart-clots.

Abdomen.—The liver was enlarged, of firm consistence, and full of venous blood, but otherwise normal. The spleen was much enlarged, of firm consistence, and its trabeculæ were unusually distinct. Its peritoneal covering was opaque, bluish-white in color, somewhat thickened, and presented a slightly corrugated appearance. There were also a great number of very small whitish granules of lymph, that had been a long time organized, firmly attached to the peritoneal coat on its free surface. The kidneys were rather firmer in consistence than natural, and congested with venous blood. The veins coming from the stomach and intestines were all full of blood. The lacteals were full of chyle and presented a beautiful appearance. Stomach was contracted, and contained some food undergoing digestion. Small intestines contained chyme. The coats of the stomach and intestines exhibited venous congestion, but in other respects they were normal. No tubercles were found in any organ.

Comments.—The immediate cause of death in this case appears to have been apoplexy, for no other hypothesis will satisfactorily account for the phenomena and anatomical lesions which it presented. Death did not begin at the heart, for the hyperæmic condition of the lungs denoted that the heart had continued to contract, and to send blood into the lungs after the function of respiration had ceased, and the blood no longer passed through the pulmonary capillaries; it did not begin at the lungs, for those organs were not primarily affected in any way; it therefore must have begun at the brain. This view is strengthened by the fact that the pupils had become widely dilated, and the face drawn to one side, as in hemiplegia from cerebral disease. Moreover, there are only two diseases of the brain which can so quickly destroy life in persons apparently well, as occurred in this case. They are apoplexy and epilepsy. This man did not have the latter disease. The undisturbed condition of the bedclothing showed that he did not have convulsions, nor pass his urine in bed, and his tongue was not bitten. He did not exhibit any of the phenomena of epilepsy, and there is, therefore, no good reason for supposing that he had that disease. On the contrary, the occurrence of an apoplectic stroke would fully account for all the phenomena which attended his death: for the sleep-like appearance of his body, the hemiplegia, and the paralysis of the function of respiration occurring before paralysis of the heart took place. There is, therefore, no good reason for doubting that he died of apoplexy.

The hypertrophy of his heart, which had been induced by insufficiency of the aortic valves, was, in all probability, the predisposing cause of the apoplexy. The disturbances in the cerebral circulation which it occasioned, would tend to impair the nutrition of the brain, and render it more liable to have its functions suddenly arrested.

This man's habits, as to drinking, were good, and therefore his brain was found to be remarkably free from disease, at the autopsy. It is probable, however, that there was a state of cerebral anæmia, especially on the left side of the organ, which failed to receive sufficient attention at our hands; and it would be more likely to be overlooked from the fact that the examination was hurried from want of time, and was made several years ago when, under the influence of the prevailing doctrines, cerebral hyperæmia received much more attention than cerebral anæmia, as a cause of death.

Furthermore, it is not improbable that in this case the anæmia of the cerebral substance, that is, the efficient cause of the apoplectic coma, was due to spasmodic contraction of the cerebral blood-vessels, tonic in character, and vaso-motory or reflex in origin. If this hypothesis be correct, the disease in this case bears a strong relationship to epilepsy; for the condition of the cerebral circulation appears to be the same in both of these, and the objective symptoms are alike in both, except the convulsive movements with which epilepsy is usually attended. It is, therefore, probable that this case belongs to the same category as those of nervous apoplexy, for an account of which see Chapter VII.

Case XVIII.

Senile bronchitis and emphysema of the lungs; sudden death from apoplexy; autopsy.

Mary Eitcher, a Jewess, aged 62, but looking much older than that, after being in poor health for some time, suddenly became comatose as she was in the act of taking her seat at the breakfast table, on the morning of March 17th, and quickly died. There was no physician in attendance.

Autopsy, by the author, thirty hours after death.—The subcutaneous adipose tissue was abundant. The post-mortem rigidity was wanting.

Head.—The arachnoid membrane was opaque, to a considerable extent, from serous infiltration. The brain was much shrunken from atrophy, and its substance was softer than natural. It was not congested, and there was no excess of serum in the ventricles, or in the meninges, above that which was required to compensate for the cerebral atrophy.

Thorax.—The bronchiæ were inflamed, and contained an abundant quantity of muco-purulent matter having a dark color. Both lungs exhibited vesicular emphysema, and were also extensively infiltrated with pigmentum nigrum. The right lung was much congested and œdematous throughout its whole extent. The left lung also was congested, but not in so great a degree. The heart was fatty. The coronary arteries were ossified. The semilunar valves of the aorta and pulmonary artery were atrophied to such an extent that circular perforations of considerable size were found near their bases and free margins. The base of the mitral valve was thickened by an atheromatous deposit. The lining membrane of the aorta also was atheromatous, commencing a short distance above its root. The heart contained no coagula.

Abdomen.—The liver was enlarged. The gall-bladder contained several calculi. The mucous membrane of the gall-bladder was inflamed where it had been in contact with the calculi.

Comments.—In this case apoplexy was caused by old age, that is to say, it resulted from the lesions or changes in structure of the organism which are incident to an advanced period of life. Prominent among these lesions are atrophy and softening of the brain, certain changes in the structure of the heart and its valves,—such, for example, as hypertrophy and fatty degeneration of the former, and atrophy, atheroma, and ossification of the latter,—and certain diseases in the coats of the arteries, such as inflammation, fatty degeneration, calcification, and ossification. When the substance of the brain has become much degenerated, and the organs for the circulation of the blood are greatly disordered, as we find them to be in advanced age, we can readily understand that disturbing causes which otherwise would be trivial and of no importance, may produce sudden anæmia of the substance of the brain, sudden paralysis of its nerve-fibres and ganglion-cells, that is, sudden arrest of its functions, or the symptoms of apoplexy; and if the medulla oblongata also happens to be involved in the cerebral anæmia, the respiratory movements are suspended, and the coma ends in death, as it did in the case of the old woman whose history we have just related.

She had been in feeble health for some time, not only because of the general state of atrophy and disease mentioned above, but also from the presence of senile bronchitis, and senile emphysema of the lungs. There was no physician in attendance, however, and thus it becomes probable that in these respects her symptoms were no worse than usual on the morning when she died; but a hard fit of coughing might readily have excited the apoplectic stroke, and so determined the fatal moment.

It is not improbable that in this case, as in the preceding one, the stroke of apoplexy was, in reality, due to a spasmodic contraction of the cerebral blood-vessels, tonic in character, and vaso-motory in origin; the spasm itself being excited by irritation reflected from some distant part, as the pulmonary organs, for example, which were found diseased in the last case.

Case XIX.

Sudden death from congestive apoplexy; no autopsy allowed.

J. H. G., a professional man of eminence, aged 45, fleshy and corpulent in habit, face full and red, addicted to over-indulgence in luxurious food and stimulating drink, suddenly fell to the floor in an unconscious state, while putting on his clothes, about 7 o'clock A.M. He had slept well over-night, and seemed to be in good health and spirits. Just after getting out of bed, and a few minutes before the attack, he made some playful remarks to his wife. I saw him about nine o'clock. He was then in deep coma; his face was flushed and looked rather bloated; his pupils contracted; his carotids throbbing; his skin warm and moist; his pulse frequent, rather feeble and somewhat irregular; his breathing stertorous with puffing of the cheeks attending each expiration; there were some slight spasms of the muscles of the face, and sometimes of the hands; but there was no facial deformity, nor any other evidence of hemiplegia. It was said that his symptoms had remained about the same ever since the attack. He had had no general convulsions. He was bled in the arm without any benefit. A stimulating enema was administered, but it also did no good. Sinapisms were applied to the epigastrium, to the region of the spine, and to the extremities, but he grew worse, he got rattles in the throat from the occurrence of pulmonary œdema, his coma became more profound, his pulse grew faster, weaker, and more irregular, his face pale, and he died about two o'clock P.M., seven hours subsequent to the attack. No autopsy was allowed.

Comments.—The symptoms afforded no evidence that cerebral hemorrhage had occurred. If a post-mortem examination of his body had been made, it is probable that lesions of the brain and other organs would have been discovered, similar in character to those which were found in case No. IV., the subject of that history being also plethoric, corpulent, and a high liver.

CASE XX.

Congestive apoplexy; death twenty hours after the stroke occurred; no autopsy.

Mr. W., a commissary of subsistence, aged about 40, of full habit, face full and red, person inclined to corpulence, a free liver, and subject to almost constant plethora from his mode of life, was stricken down by apoplexy on Saturday morning, April 26th, about six o'clock, while supervising the issue of rations from his store-house. A few minutes afterward (not more than ten) I saw him, and learned that the attack had come on suddenly and without warning. He had fallen suddenly to the floor in a state of insensibility, from which his attendants could not rouse him. I found him in deep coma, that is, utterly insensible; his respiration slow, deep, and snoring; his face dark-red, and turgid, from hyperæmia; his carotids beating noticeably; his conjunctivæ injected, and pupils contracted but symmetrical; his pulse slow, full, and strong. His countenance was not distorted, and there was no sign of hemiplegia or any other form of local palsy. Twitchings of the facial muscles were occasionally seen, but there were no general convulsive movements.

Treatment.—Pouring cold water on his head, the trunk being raised up and held in a semi-sitting posture the while, was employed without success. Then general blood-letting was resorted to, but without any benefit. On the contrary, it appeared to make his pulse more frequent and weak. Calomel was administered as a purgative. Sinapisms were applied to his epigastrium, nucha, thighs, ankles, wrists, and kept on a long time. Tinct. rad. aconiti and infus. ipecacuan. were also administered. His bowels failing to act with sufficient promptitude, an enema was given. Wet cups were applied to his nucha. Finally, as every thing else had failed to benefit him, the lower half of his body was placed in a hot bath, and cold water was poured on his head at the same time. This also did no good. Mr. W. was a great favorite with every one who knew him; and no pains nor toil were spared to save him. But, in spite of all that we could do, he sank from coma into carus, and died about two o'clock on Sunday morning, April 27, twenty hours after the stroke occurred. No autopsy.

Comments.—This case also presents us with a typical instance of apoplexy in the ancient and clinical sense of the term. This man suddenly lost consciousness and the power of voluntary motion. He also sank into what appeared to be a very deep sleep, which ended in death after many hours. But from first to last there was no distortion of his face, no appearance of paralysis in the muscles of his arms or legs on either side of the body, and no sign whatever of hemiplegia. This observation makes it pretty sure that no cerebral hemorrhage of moment occurred, and that the apoplectic phenomena were not due to that cause. If an autopsy had been held we doubtless would have found strongly-marked hyperæmia of the brain and its membranes. We would have found those structures as turgid with blood as were his face and eyes. We would have found substantially the same post-mortem lesions as we have described in a considerable number of analogous cases.

Toward the end of this man's sickness the cerebral paralysis deepened, and the powers of his system gradually failed; the pupils became symmetrically dilated. From this circumstance we infer that some serum was effused at the base of his brain, and in the meshes of the subarachnoid connective tissue generally. He had hypertrophy of the heart, and this pathological state afforded one reason for our resorting to blood-letting for the purpose of relieving his head.

CASE XXI.

Congestive apoplexy occurring in a female aged about fifty; recovery.

On a Sunday afternoon some years ago I was called in great haste to the sister of a Mrs. W. The messenger seemed badly scared—said the lady was in a fit and they thought her dying. I found her down with apoplexy, and saw at a glance that she was quite corpulent and considerably past the middle period of life; that her face was swollen and deep-red from hyperæmia, that her carotids were throbbing violently, and that her respiration was slow, deep, labored, and stertorous. She was in profound stupor and could not be roused, her pulse was slow, full, and regular, her pupils contracted, and conjunctivæ injected. She had been attacked suddenly and without warning about twenty minutes before my arrival. She had suddenly sunk back in her chair in an insensible state, with flushed face, while conversing with some neighbors. They experienced much difficulty in placing her on a bed which stood in the room, because of her great weight and helpless condition. They loosened her clothes, but did not remove them. She had continued to lay where placed on the bed, without moving, and just the same as I found her, they said. She had not had any convulsions. There was no facial distortion, and no symptom of hemiplegia, or any other form of muscular paralysis.

I afterward learned, further, that she was unmarried, and about fifty years old, that she had passed the change of life, that recently she had been acquiring flesh very fast, that formerly she had indulged in the use of opium to great excess for years, but that recently she had abandoned it either entirely or in great measure, that while she indulged in opium-eating she kept pale and thin, and that she began to improve in color and flesh as soon as she conquered her evil habit.

Treatment.—I corded her arm and tried to bleed, but failed to find the vein because her arm was so fat. The symptoms were very threatening. Her countenance was enormously congested, her breathing very laborious, deeply stertorous, and rapidly growing worse. I expected every moment to see her mouth become distorted and drawn to one side from facial paralysis, and thus hemiplegia from cerebral hemorrhage set in. The case appeared so urgent that I did not stop for trifles. I propped up her head with pillows, as she lay in bed, as soon as possible, and began to pour cold water upon it from a pitcher held at a little height, in a small, steady stream. As fast as one pitcher was emptied another was handed me by a young lady who had kept her senses in the midst of the general panic. Soon I stood up on a chair, and thus was enabled to pour the water from a still greater height. The height of stream employed varied from six inches to two feet. Under this treatment her face gradually assumed a less congested and more natural appearance, and her breathing became less stertorous. After some minutes she began to corrugate her forehead, or scowl, as if she felt uncomfortable. Soon afterward she began to move her head as if she wished to avoid the stream of water. In about twenty minutes she opened her eyes and asked what was the matter. The water-pouring was then stopped. Her consciousness was now completely restored, and her appearance quite natural. She was supplied with dry clothing and placed in a dry bed. Pil. cathartic comp. No. 3 were administered as a purgative, quietude was enjoined, and a milk diet allowed. Her case gave no further trouble, as her recovery was speedy and complete.

Comments.—This woman had probably acquired a predisposition to the occurrence of apoplexy by eating opium. The abuse of narcotics as well as alcoholic intemperance is generally considered to hold an important place among the causes of this disease. Opium exerts a powerful influence on the capillary circulation of the blood, and the vaso-motor system of nerves, and by this means, when habitually taken, doubtless modifies the nutrition of the brain and ganglia of the sympathetic system to a considerable extent, as well as that of other organs and tissues. In acute poisoning by opium the brain is not congested but anæmic, as already mentioned, and as will be again shown in another place.

Case XXII.

Apoplexy congestive (so-called) with symptoms of hemiplegia; recovery.

Mr. McC., aged about 60, employed as head-salesman in a large retail store for furnishing and dry goods generally; work laborious and confining; complexion rather pale, and hair very white, health generally good, accustomed to take alcoholic drinks, especially brandy and water, freely, and sometimes to excess; had an apoplectic fit in the store about four o'clock P.M., and a few minutes afterward I saw him. He lay in a deep stupor from which he could not be roused, his face was somewhat flushed, his pupils contracted, his mouth drawn to one side from facial paralysis, his respiration slow, deep, and snoring, his skin warm and moist, his pulse slow, regular, and soft. There were no convulsions, and had been none from the start. The coma occurred suddenly. I had him taken home in a carriage, and accompanied him. He was carried up-stairs to his room and placed in bed. His symptoms then were about the same as when I first saw him. Ordered ice to be kept on his head, to take tinct. rad. aconiti gtt. j. every four hours, and to have his bowels moved by enema. Sinapisms had already been freely applied to his epigastrium, legs, thighs, and arms.

He regained consciousness on the second morning afterward. It was now found that, in addition to the one-sided palsy of his face, he had the usual symptoms of hemiplegia. He had no heart-disease. The aconite was continued; his head was kept wet with cold water instead of ice; a blister was applied to the back of his neck, and a milk diet with beef-tea was allowed. Under this treatment he improved with great rapidity, and at the end of a fortnight all the objective signs of paralysis had disappeared. He soon afterward recovered perfectly the use and strength of his paralyzed limbs.

Comments.—In this case the symptoms of apoplexy and hemiplegia could not have been due to cerebral hemorrhage nor to cerebral softening, for if they had been, recovery in the manner and to the extent mentioned above could not have taken place. They must therefore have been occasioned by some functional disturbance of the brain, some temporary anæmia of the ganglion-cells and nerve-fibres of one lateral half of the organ.

We have related in this chapter seventeen cases of apoplexy which belong to the so-called congestive and serous varieties of the disease. They occurred in all classes of society, from the respectable to the low and debased. In respect to age the following is a summary: *One* case occurred between 20 and 30; *four*, between 30 and 40; *six*, between 40 and 50; *four*, between 50 and 60; *two*, between 60 and 70: the *youngest* was 22, the *oldest* 62. In respect to special etiology *alcoholic intemperance* occupies the front rank, for it was a prominent cause of the disease in ten of the seventeen cases, and probably in one or two more than that. Four subjects were *aged*, although prematurely so. Five subjects were addicted to the *pleasures of the table.* One had *emphysema of the lungs and hypertrophy of the heart*, and one, *hypertrophy of the heart with insufficiency of the aortic valves;* but a *moderate amount of cardiac hypertrophy* was found in several other cases. The habit of *opium-eating* was the predisposing cause in one case. *Strong mental emotion* was the exciting cause of the disease in two cases. *Concussion or commotion of the brain* was the exciting cause in one case, and perhaps more.

A perusal of these cases of apoplexy further shows that the boundary between the congestive and serous varieties of the disease is not well defined; that they are seldom, if ever, distinguishable during life, and that oftentimes they are not separable even on examination after death. Congestion or hyperæmia of the brain and its membranes, and œdema of the brain and its membranes, are pathological states of those structures which are quickly and easily convertible the one into the other, by the simple transudation and absorption of serum. This transudation of serum often takes place in the brain and lungs during the act of dying, and probably after death also. (See comments on Case VI.) Case IV. shows very clearly that the so-called congestive and serous varieties of the disease are not clinically distinct from each other.

The symptoms were generally well marked; but in some of the cases they were very striking, so much so as to constitute what may, with propriety, be called *apoplexia fulminans*, *apoplexie foudroyante*, or *thundering apoplexy.* A symptom of great importance was present in all the cases that were seen by the author during life, namely, throbbing of the carotid arteries. This symptom, when it occurs in cases where the heart and carotids themselves are not diseased, and where the other arteries do not throb, denotes that some considerable obstruction to the passage of blood through the brain exists, as already pointed out by Niemeyer. This symptom was, doubtless, also present in the other cases of apoplexy which we have related.

In thirteen of the seventeen cases above-mentioned a post-mortem examination was made. In some of them the morbid appearances or lesions of the brain and its membranes were very striking, and in nearly all of them they were very significant. The substance of the brain was obviously diseased in almost every instance. It was more or less atrophied in every case but two. In some of them the cerebrum was much shrunken. In five of them, all intemperate, the brain-substance was decidedly firmer than natural, or indurated, and in four, mostly aged people, it was softened. In all, or nearly all, these cases the blood-vessels of the brain-substance were found to be dilated in consequence of chronic congestion. In several of them this vascular dilatation was very strongly marked, so much so as to give thin slices of the cerebrum a "cribriform" or "sieve-like" appearance. Again, there was hyperæmia and œdema of the brain and its membranes in a large majority of these thirteen cases. In some of them—for example, Cases I., II., and XII.—the hyperæmia largely predominated; in others—for example, Case VI.—the œdema. When the substance of the brain is markedly or highly œdematous, the surface of a section is pale, moist, and shining. Œdema of the meninges gives the visceral arachnoid a pale gelatinous appearance. But neither hyperæmia nor œdema of the brain and its membranes is likely to make much show at the post-mortem examination, unless the brain itself is atrophied, or diminished in size, enough to allow distention of the blood-vessels, and transudation of serum into the perivascular spaces and subarachnoidean connective tissue, to simultaneously occur. We have already remarked that cerebral congestion does not produce apoplectiform phenomena in cases where the brain is healthy, or, in other words, that it requires something more than cerebral hyperæmia to produce apoplexy, and this view is supported by these autopsies.

The proximate or efficient cause of the apoplectiform phenomena in each of these seventeen cases was, in all probability, a deficient supply of freshly oxygenated blood to the nerve-fibres and ganglion-cells of the brain. In one class of these cases—for example, Cases I., II., and III.—this appears to have been due to suddenly occurring vaso-motor paralysis of the cerebral blood-vessels and stagnation of their contents. In such cases fresh arterial blood fails to enter the brain-substance because the effete venous blood does not pass out of it. This form of cerebral hyperæmia is usually associated with a state of general venous congestion or overfulness of the systemic veins. The blood stagnates in the cerebral vessels in such cases, firstly, because of this distended condition of the veins in general; and, secondly, because the contractility and elasticity of the walls of the cerebral blood-vessels themselves have been destroyed by paralysis of their vaso-motor nerves.* That the blood really stagnates in the veins in the cases of congestive apoplexy, which belong to this category, is proved by the fact that, not unfrequently, on performing venesection in such cases, the blood runs very slowly at first, or does not start at all. Some years ago, I had occasion to employ general blood-letting, at Bellevue Hospital, in a case of congestive apoplexy where the symptoms were very urgent, and this proceeding seemed to be strongly indicated. I opened the median cephalic vein of the right arm, but only a few drops of blood came away. Then I opened the median cephalic vein of the left arm, but with the same result. As a dernier resort I opened the anterior branch of the right temporal artery, and with complete success. After the blood had flowed a short time the patient's breathing improved. The livid hue of his countenance disappeared, and in a short time longer his consciousness was completely restored. The bleeding from the temporal artery was now stopped in the usual way, that is, by completing the division of the vessel and applying pressure. But I was then surprised to find that he was bleeding quite profusely from each of the incisions in his arms. This hemorrhage, however, was readily stayed by dressing each wound with a compress and appropriate bandage. In this case the blood in the veins was so completely stagnant that it could not flow out when they were first opened, but as the bleeding from the temporal artery progressed the general venous hyperæmia subsided, and the blood in the veins got in motion again. We may here remark that this patient, although relieved of his apoplectic symptoms, died a few days afterward of chronic alcoholism.

In another important class of these seventeen cases, the supply of arterial blood to the histological elements of the brain was suddenly arrested by the occurrence of cerebral œdema, that is, by the sudden transudation of serum into the perivascular spaces of the brain-substance, in such quantity as to compress the capillaries, render them anæmic, and arrest the functions of the nerve-fibres and ganglion-cells of this substance. Case VI., affords an excellent illustration of this form of apoplexy. It is probable that cerebral œdema also played an important part in the production of apoplectic coma in Cases III., IV., V., VII., VIII., IX., XI., etc. When the brain is much atrophied, a state of anæmia of the nerve-fibres and ganglion-cells, the product of cerebral œdema, is compatible with a state of hyperæmia of all the larger vessels belonging to the brain and its membranes, or, in other words, in such cases, the large vessels of the brain and its membranes may be distended with blood, while the brain-substance itself is anæmic from compression of its capillaries by the serum that is effused into the perivascular spaces.

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* Niemeyer, while enumerating the causes of cerebral hyperæmia, gives much prominence to paralysis of the vaso-motor nerves of the cerebral blood-vessels. He says: "Physiological experiments show that if the cervical portion of the sympathetic nerve be divided, the vessels on the corresponding side of the head become dilated. The cerebral vessels appear to be similarly affected by the abuse of spirituous liquors, by some poisons, as well as by great emotions and excessive mental activity. I would particularly call attention to the last cause, as I have frequently seen dangerous hyperæmia of the brain after too prolonged mental labor, which resulted fatally from the occurrence of œdema. We can hardly give any other explanation for these cases than that the walls of the vessels are paralyzed by the above influences, their calibre dilated, and the supply of blood consequently increased." (Vide *Text-book of Practical Med.*, Vol. II., p. 158, first Am. ed.) See also the comments on Case III. in this connection.

It is probable that in some, perhaps in a large share, of these seventeen cases of apoplexy, the supply of freshly oxygenated blood was withheld from the nerve-fibres and ganglion-cells of the brain through the joint operation of vaso-motor paralysis of the cerebral blood-vessels and stagnation of their contents on the one hand, and œdema of the brain, or sudden transudation of serum into the perivascular spaces of the cerebral substance on the other, or, in other words, that in some, perhaps in many, of our cases, the apoplectic phenomena were due to the presence of both hyperæmia and œdema of the brain.

For purposes of comparison and contrast, we have also related in this chapter one case of fatal coma, due to epileptiform convulsions, (Case XIII.,) two cases of eclampsic convulsions and coma, due to the uræmic intoxication of Bright's disease, (Cases XIV. and XV.,) one case of lethargy and coma also due, in all probability, to poisoning of the blood with urea, (Case X.,) and one case of convulsions and coma resulting from softening of the brain, (Case XVI.) Most of these cases will be referred to again when we come to speak on the diagnosis of apoplexy.

The next case is believed to be an instance of poisoning by opium or morphia. It is introduced here for the purpose of contrasting its phenomena and post-mortem lesions with those of apoplexy. It has already been referred to in Chapter II.

Case XXIII.

Suicidal poisoning from opium or morphia; death; autopsy; substance of brain anæmic, etc.

Mary Quinn, aged 23, a house-maid, had been in a desponding state of mind for some time, owing to disappointment in love. She had several times been heard to wish for death. Her bodily health, however, was good. Another servant found her in a state of insensibility between four and five o'clock on the morning of Tuesday, August 17th. Dr. Maxwell was called, and saw her about seven o'clock. She lay in a state of complete insensibility, with contracted pupils. Her respiration was very slow, only six or seven times per minute, irregular, and stertorous. There were rattles in her throat and chest, and the power of swallowing had ceased. Her pulse was one hundred and thirty-two. Her surface was warm, her face turgid with blood, and lips livid. The stomach-pump was used; and bleeding from the arm was employed to relieve the general venous congestion. Dr. White also saw her. Both doctors thought from her appearance that she had lain comatose most of the night. She grew progressively worse, and died about midnight.

Autopsy, by the author, August 18th, twelve hours after death.—Face pale, pupils dilated, (equally,) rigor mortis wanting. Body that of a robust servant-girl.

Head.—Skull thick. No blood or serum escaped on removing the skull-cap. Some old bridle-shaped adhesions between the two surfaces of the arachnoid were found on the convex surface of both hemispheres of the cerebrum. The sinuses of the dura mater and veins of the pia mater were full, but not distended with blood. The substance of the brain was not congested but, on the contrary, exsanguinated. The puncta vasculosa were few and small. The brain-substance was everywhere pale, but its consistence was normal. The ventricles were empty, and there was no œdema of the meninges.

Thorax.—The lungs collapsed well on opening the chest. The right lung was bound to the diaphragm throughout almost all its base by firm, old cellular adhesions. It was also adherent to the chest posteriorly. Each lung contained more than the normal quantity of blood, and was œdematous, especially in its upper lobe. The heart was rather fatty on its exterior, but otherwise normal. Foramen ovale closed. The blood was fluid in all parts of the body.

Abdomen.—Stomach distended with gas. It also contained about ten ounces of undigested and partially digested food. Its mucous membrane exhibited a pale color throughout, with some mammillations on the great curvature toward or near the pyloric extremity of the organ. We did not detect the odor of laudanum or opium. Spleen glued to the diaphragm by some old adhesions, but otherwise natural. Liver, kidneys, and intestines normal. Uterus unimpregnated. Ovaries (principally the right) exhibited corpora menstrualia of various dates.

Comments.—Was this a case of poisoning by opium or its active principle morphia? The physicians who saw the patient during life were of the opinion that it was. In this opinion the author fully concurs. Her death was, in all probability, due to the taking of some kind of poison, for robust young women of good habits, who are as free from disease of the heart, blood-vessels, and brain, as she was, are not apt to die as suddenly as she did from the operation of natural causes alone. If she took poison, it was some narcotic; and it must have been some preparation of opium, for of all the narcotics this is the only one which produces the phenomena which she exhibited, namely, strong contraction of the pupils, and extreme slowness of the respiratory movements—only six or seven per minute. Her symptoms were precisely those which are produced by opium or morphia poisoning and by nothing else, and therefore we have no doubt that she died in consequence of taking such a poison.

The most noteworthy feature presented by the autopsy was the exsanguinated or anæmic condition of the brain-substance. We have already referred to Mushet's case of poisoning by laudanum in which "the brain presented a very pale, almost anæmic appearance," (*op. cit.* p. 162.) Does opium, when taken in poisonous doses, produce death by inducing anæmia of the substance of the brain?

We have, in this chapter, discussed the clinical phenomena and post-mortem appearances which are found in cases of simple apoplexy, at great length, and with considerable reiteration. For doing this our apology is, 1. that some persons of prominence in the profession have doubted whether the congestive and serous varieties of apoplexy ever occur; 2. that a recent author (Vide *A Practical Treatise on Apoplexy, etc., by William Boyd Mushet,* M.B. London, 1866) strenuously contends against admitting simple apoplexy to be a substantive disease; and 3. that the subject possesses very great importance.

CHAPTER V.

ON HEMORRHAGIC APOPLEXY OR APOPLECTIFORM CEREBRAL HEMORRHAGE, MENINGEAL APOPLEXY, AND CEREBRAL HEMORRHAGES IN GENERAL, TOGETHER WITH HÆMATOMA OF THE DURA MATER.

Definition.—*Varieties* of Cerebral Hemorrhage.—The phenomena vary considerably in different cases, and the varieties of this affection are several in number.—1st Variety: In it there are no apoplectic symptoms, no loss of consciousness, no coma, nor carus; but hemiplegia is generally present; considerably more than one third of all the cases belong to it.—2d Variety: In it loss of consciousness or coma occurs, but the symptoms come on gradually, that is, they are not developed with sufficient suddenness to constitute an apoplectic fit; the patient recovers, but remains paralyzed for the rest of his life; this variety also is often met with.—3d Variety: In it the attack soon produces coma which passes away in a short time as in the last variety; but after a few hours consciousness again begins to disappear, and finally is entirely lost; it does not return, and the patient dies comatose.—4th Variety: In it the attack begins with the symptoms of hemiplegia, but the patient is perfectly conscious; later, sopor, coma, and carus occur, and death from paralysis of the medulla oblongata in the end ensues; it often occurs.—5th Variety: In it the hemorrhage occurs during the progress of a fit of epilepsy or eclampsia; two or more cases of this sort will be related in this chapter.—6th Variety: It is the so-called *apoplexie foudroyante* or thundering apoplexy of authors, and is the only form of cerebral hemorrhage which is attended with the symptoms of "stroke" in the proper sense of the word; it is apoplectiform cerebral hemorrhage or hemorrhagic apoplexy, and is comparatively seldom met with.—The last two varieties will claim much of our attention.—*Case* XXIV. Fit of apoplexy and death in ten or fifteen minutes; autopsy; much coagulated blood found in the arachnoid cavity and in the ventricles; brain-substance and meninges considerably exsanguinated, etc.—*Case* XXV. Puerperal eclampsia and death in less than three quarters of an hour; autopsy; very copious and extensive hemorrhage into the brain; brain-substance and membranes found to be much exsanguinated, etc.—*Case* XXVI. Epileptiform convulsions occurring in an intemperate subject; the second paroxysm ended in death; autopsy; found very extensive meningeal hemorrhage, also ventricular hemorrhage; brain atrophied, indurated, and congested; meninges congested and œdematous; the lesions of the brain and other organs which are produced by alcoholic intemperance enumerated.—*Case* XXVII. A man found dead in the street; symptoms unknown; autopsy; found meningeal hemorrhage of a profuse character; also some ventricular hemorrhage; substance of brain anæmic or exsanguinated; medulla oblongata compressed by the extravasation; exciting cause of the hemorrhage probably concussion of the brain; predisposing cause, intense cerebral congestion.—*Case* XXVIII. A man died suddenly of coma soon after having been beaten; autopsy; found profuse meningeal hemorrhage; slight ventricular hemorrhage; brain atrophied and congested; kidneys strongly congested; alcoholic lesions of the brain beginning to show themselves.—*Case* XXIX. Sudden death attended probably by epileptiform convulsions; autopsy; found very copious meningeal hemorrhage; substance of the brain markedly anæmic or exsanguinated; symptoms of epilepsy. —*Case* XXX. Eclampsia occurring in a new-born child; death; autopsy; found extensive meningeal hemorrhage, the result, in part at least, of injury.—These seven cases all come under the general title of apoplectiform cerebral hemorrhage.—Résumé of these cases; in only one of them was the loss of consciousness known to have been due to the extravasation, while in the other six it was ascribable to either epileptiform convulsions or to the shock of injury; thus our experience supports Trousseau's views; in six cases the hemorrhage was meningeal; in but one into the substance of the brain; the subjects were for the most part remarkably young; in five of the six adults the kidneys were considerably diseased; this relationship of renal disorder to cerebral hemorrhage something more than mere coincidence.—Our cases show that some of the views of authors concerning meningeal hemorrhage are erroneous; also that meningeal hemorrhage is apt to prove quickly fatal, and that cerebral hemorrhage generally produces anæmia of the brain-substance; some exceptions, however, are mentioned and explained. The circumstances stated under which cerebral hemorrhage may produce apoplectic coma, properly so-called.—*Etiology of Cerebral Hemorrhages in General;* views and discoveries of Drs. Charcot and Bouchard; miliary aneurisms of the minute intra-cerebral vessels; Prof. Béhier confirms this discovery; fatty or granular degeneration of the capillaries of the brain as a cause of cerebral hemorrhage; also an atheromatous condition of the cerebral arteries and intra-cranial aneurisms in the ordinary sense of the term; increased tension of blood in the cerebral arteries; certain morbid states of the blood itself; but cerebral hemorrhage is especially apt to occur when several of these causes coöperate.—*Anatomical Appearances produced by Cerebral Hemorrhages in general;* appearances pertaining to capillary hemorrhages; also to hemorrhagic clots; the parts of the brain in which these extravasations are found, and the relative frequency; the contents of a hemorrhagic cavity, and the changes thereof described; apoplectic cysts, apoplectic cicatrices, and apoplectic abscesses; anæmia and other alterations in the parts of the brain untouched by the hemorrhage described; also age of the clot, and how it is determined; blood-crystals in the cavity; when several hemorrhagic cavities are found, they are generally of different ages.—*Symptoms and Course of Cerebral Hemorrhage,* illustrated by brief sketches of six cases, given by Trousseau; it generally, but not always, occurs unexpectedly or without warning; the warning symptoms enumerated; on hemiplegia as a symptom of cerebral hemorrhage; its relation to extravasations in different parts of the brain; hemorrhage in the cerebrum, pons, medulla oblongata, and cerebellum; seven eighths of all cerebral hemorrhages are located in the cerebrum; vomiting, contraction of the pupils, slowness of the pulse, and throbbing of the carotids considered as symptoms.—*Case* XXXI. Extravasation of blood in the pons Varolii terminating in recovery; intellect unaffected; symptoms related.—*Case* XXXII. Hemorrhage in the left crus cerebri; sudden paralysis of right side of body; intellect unaffected; death from broncho-pneumonia; small coagulum in left crus cerebri, etc.—Symptoms of sanguineous effusion in the substance of the cerebral hemispheres enumerated: 1. Mental; 2. Sensorial; 3. Motorial.—Symptoms of hemorrhagic effusion into the ventricles: 1. Mental; 2. Sensorial; 3. Motorial.—The facial palsy of cerebral hemorrhage; its peculiarities.—Hemiplegia not characteristic of cerebral hemorrhage; it is produced by other disorders of the brain; they are enumerated.—On the secondary encephalitis and fever which are produced by cerebral hemorrhage.—*Prognosis of Cerebral Hemorrhage;* some bad prognostics mentioned; the practitioner should be guarded in stating the prognosis; circumstances which should be remembered in framing the prognosis.—*Treatment of Cerebral Hemorrhage:* 1. Before the attack; when warning symptoms are present, the therapeutic indications should be carefully fulfilled, and the way is pointed out; 2. During the attack; venesection should generally not be practised; the therapeutic indications stated; occasionally, however, it is advisable to bleed; the symptomatic indications for it given; the symptomatic indications for the use of stimulants also given; 3. After the attack; attention to diet; use of laxatives, and cold applied to the head;

venesection always superfluous and even dangerous in this stage; use of counter-irritants, etc.—*Concerning Hæmatoma of the Dura Mater;* definition; etiology; anatomical appearances; symptoms and course; treatment.—Opinions of Charcot, Vulpian, and Lancereaux on this disease.

Definition.—By the term cerebral hemorrhage we mean an extravasation of blood into the substance or into the ventricles, or upon the surface of the brain, (encephalon,) or either of its four principal divisions, namely, the cerebrum, cerebellum, pons Varolii, and medulla oblongata. The term encephalic hemorrhage would be more critically accurate than the term cerebral hemorrhage. Extravasation of blood upon the surface of the brain is often called meningeal hemorrhage, because the effusion is, strictly speaking, located in the meninges of the brain in such cases. But, inasmuch as meningeal hemorrhage derives its importance solely from the deleterious influence which it exerts upon the brain-substance, we shall consider it merely as an important variety of encephalic or cerebral hemorrhage.

Varieties of Cerebral Hemorrhage.—The phenomena which are produced by cerebral hemorrhage vary considerably in different cases. They depend upon the part of the brain in which the extravasation occurs, the size of the clot itself, the rapidity with which the blood forming it is effused, and the amount of injury done to the nerve-fibres and ganglion-cells. A small clot in the subarachnoid connective tissue occasions headache, some confusion of thought, and but little else in the way of cerebral disturbance; while in the medulla oblongata it suspends the functions of that part of the brain, paralyzes the respiratory nerves, arrests the respiratory movements, and occasions death. A small clot in either of the superior motor ganglia of the cerebrum, the corpora striata, and the optic thalami, occasions paralysis on the opposite side of the body or hemiplegia, but without loss of consciousness; a large clot in the same situation produces not only hemiplegia, but coma also. When we consider that hemorrhage may occur in any part of the brain, that the clot may be single or multiple, and that it may vary in size from a pin-head or pea to a large pear or orange, we must expect to find that this disease presents many varieties in its clinical phenomena as well as in its anatomical features. The cases of cerebral hemorrhage may be classified under several different heads, according to the symptoms, course, consequences, and post-mortem appearances which they present, that is, according to the size and number of the clots, their different seats, the rapidity of their formation, and the severity of the consecutive inflammation in the surrounding brain-substance. The following are the chief varieties:

1. In the cases belonging to this class the attack is not attended with apoplectiform phenomena. The patient does not have coma. He loses consciousness either not at all, or only momentally, or partially and incompletely. He is stupid and confused in his mind for a time, in consequence

of the shock his brain has received. The symptoms are generally those of hemiplegia. There is paralysis more or less complete of one lateral half of his body, that is, of the muscles of his face, on one side, and likewise of the muscles of his upper and lower extremity on the same side. The motor palsy is accompanied with more or less anæsthesia or sensory palsy, but the latter is usually much less profound and persistent than the former. There is neither coma nor carus in the whole course of this variety of cerebral hemorrhage, unless it happens to be produced by the secondary inflammation of the brain-substance or meninges around the clots; and the subjects of it, for the most part, recover more or less completely. In these cases the clots are probably small, and the nerve-fibres and ganglion-cells are pushed apart by the extravasation, instead of being broken down and destroyed. Hence we find in such cases that as the clot becomes absorbed the symptoms of hemiplegia gradually disappear. The recovery, however, but seldom becomes perfect. Even in the most successful cases some debility almost always remains for life in the muscles of the arm and leg on the affected side. This variety of cerebral hemorrhage is of relatively very frequent occurrence. It is probable that considerably more than one third of all the cases of cerebral hemorrhage belong to it, for T. Jones has found, in forty cases of cerebral hemorrhage which proved fatal, that the consciousness was but partially or incompletely abolished in twelve instances, or about thirty per cent of them. Now, if so large a ratio obtains among the cases which ultimately prove fatal, it is obviously fair to suppose that a still larger ratio obtains among those which recover. (Vide *New Sydenham Soc. Year-Book*, 1864, p. 92.)

2. This variety also is not unfrequently met with. In the cases belonging to it, the hemorrhage quickly produces complete loss of consciousness or coma, but it does not do it so suddenly as to constitute an apoplectic fit, in the correct sense of the term, as Trousseau has conclusively shown. After the lapse of half an hour, or several hours, or occasionally not till the next day, the patient gradually awakes from his stupor; but his speech is indistinct or thick, his mouth drawn to one side, and hemiplegia is found to be present, attended with all the signs which usually belong to it. On the second or third day afterward there are fever, headache, and other symptoms of traumatic encephalitis. After these have disappeared, the patient remains for the rest of his life paralyzed on one side of his body, although the part of the paralysis which results from œdema in the vicinity of the clot goes away after a time. In such cases the phenomena are generally due to a rapidly-recurring but limited, although pretty copious, extravasation of blood into the cerebrum, and especially in the vicinity of the corpus striatum and optic thalamus. In these cases, during even the stage of unconsciousness, we may often infer from the distortion of the face,

from the relaxation of the extensor muscles in one arm and in the corresponding leg, and sometimes also from the dilatation of one pupil, which the paralyzed side is.

3. In another class of cases, where the hemorrhage probably ceases for a time and then returns, but continues slowly or moderately, the attack also soon produces loss of consciousness and the other symptoms of coma, but they pass away in a short time, as in the last-mentioned variety. We note the hemiplegia, and hope that the patient will this time escape with his life. But after a few hours consciousness again begins to disappear, and finally is entirely lost. It does not return, and the patient dies comatose. (*Niemeyer.*)

4. In this variety of cerebral hemorrhage the attack is not ushered in with the symptoms of coma. It begins with the symptoms of hemiplegia, but the patient is perfectly conscious. Later, sopor, coma, and carus, one after the other, occur, and death from paralysis of the medulla oblongata in the end ensues. In the cases belonging to this category the blood is slowly and steadily effused, but finally the clot becomes very large. This variety of cerebral hemorrhage is often met with.

5. In some cases of epilepsy and eclampsia one or more of the cerebral blood-vessels becomes ruptured during the progress of the fit, and extravasation of blood into the brain or its membranes occurs. When cerebral hemorrhage is thus produced it must generally be considered as an important and, for the most part, fatal complication of the preëxisting disease. We have already mentioned one of Trousseau's cases which belongs to this class, and shall relate in this chapter at least two other cases belonging to the same category.

6. The last to be enumerated, and the most striking variety of all, is the so-called apoplexie foudroyante, or thundering apoplexy, when it chances to be produced by cerebral hemorrhage. It generally corresponds to a rapidly-occurring and extensive extravasation, or to the coincident occurrence of several extravasations, which involve the vital parts of the brain, and is attended with the following phenomena: After some premonitory symptoms, or even without them, an apoplectic stroke in the correct sense of the term, suddenly occurs; the patient, however, does not recover consciousness, the symptoms rapidly deepen, the paralysis quickly extends to the medulla oblongata, the breathing becomes irregular, the pulse intermittent, weak, and slow, the pupils dilated, and death occurs in a few minutes. This form of cerebral hemorrhage is the only one which is really attended with the phenomena of apoplexy. It is of very infrequent occurrence, so much so that Trousseau, notwithstanding his vast experience, never met with a case of it, as we have already mentioned. We shall, however, presently relate an undoubted instance of it. We

may here remark that the so-called apoplexie foudroyante is produced much more frequently by cerebral congestion and œdema than by cerebral hemorrhage. We may further remark that the last two varieties of cerebral hemorrhage are the only ones whose phenomena correspond with or closely resemble those of apoplexy in the etymologically correct sense of the term. These two varieties, therefore, will specially claim our attention in the following pages. After what has just been said, and elsewhere fully shown, it is scarcely necessary to state again that apoplexy and cerebral hemorrhage are not synonymous terms.

We should, however, not forget that, although hemorrhagic apoplexy is a disorder of comparatively infrequent occurrence, cerebral hemorrhage and its consequences are very often met with in practice. We frequently see people going about in our streets who have incurable hemiplegia resulting from extravasation of blood in the brain. Those people mostly require the aid of crutches or canes in order to get about, and usually drag the toes of the paralyzed side after them as they walk.

CASE XXIV.

Fit of apoplexy and death in ten or fifteen minutes; autopsy; much coagulated blood found in the arachnoid cavity and in the ventricles; brain and meninges considerably exsanguinated; heart and lungs normal; liver fatty; kidneys small, hard, and granular.

Mrs. Mary Johnson, aged 49, colored, was engaged in rinsing clothes at the hydrant in "Cow Bay," on the morning of November 11, and while in a stooping posture, suddenly fell down upon her left side, as if she had been struck down by a powerful blow. She was picked up insensible by her neighbors, and carried into a room near at hand, where she died in ten or fifteen minutes, and before a physician could be called. She generally had enjoyed good health. She was reported to have had anasarca of the lower extremities several years ago, but latterly she had not been troubled with it. The attack occurred without any cry or warning, and she did not exhibit any convulsive movements. She lay senseless, motionless, and her breathing was stertorous.

Autopsy, by the author, twenty-four hours after death.—Cadaver in good flesh; post-mortem rigidity strong; extremities and other parts not œdematous. There were no bruises or other marks of violence.

Head.—Skull thick. The right temporal region was wholly occupied by a flattened clot of blood, which lay in the arachnoid cavity, and obviously depressed the brain-substance. There was also extravasated blood in the subarachnoid connective tissue, in the same situation. Coagulated blood was likewise found lying along the track of the optic nerves at the base of the cerebrum, and about the pons Varolii and medulla oblongata.

The quantity of this extravasation was abundant about the pons and medulla oblongata. The fourth ventricle was full of coagulated blood; and there were clots in the lateral ventricles also. We found a transparent serous cyst in the right lateral ventricle, attached to the plexus choroideus, having the size and shape of a small grape. The vessels of the brain and its membranes contained considerably less than the normal quantity of blood. The substance of the brain was paler than natural, and the puncta vasculosa were few and small. Its consistence was normal. But little fluid, whether blood or serum, escaped while we were opening the head and examining the brain. We failed to discover the source of the hemorrhage.

Thorax.—The lungs were entirely free from adhesions, and normal in every respect. The heart also was natural.

Abdomen.—The liver was large, and on its surface were several spots having a dirty yellowish-brown color, resembling that of dried leaves, supposed to be due to fatty degeneration. These spots all dipped down into the substance of the organ to varying depths. The spleen was natural. The kidneys were paler in color, and smaller in size than natural. They had a firm consistence, with a mottled and granular appearance. The cortical substance, or secreting portion, of each organ, was much thinner than natural. The stomach was well contracted, but not empty. The folds of its mucous membrane had a reddish color, but no abnormity of its thickness and consistence was noted.

Comments.—This was undoubtedly a case of apoplectiform cerebral hemorrhage. The blow which struck this woman down to earth and destroyed her life was delivered not by the hand of violence, nor by a fit of epilepsy, nor by the convulsions of eclampsia; but by the spontaneous rupture of some cerebral blood-vessels, and the extravasation of blood in large quantity upon the brain. The sudden extinction of her consciousness and sensibility was due in part to the shock, but mainly to the anæmia of the nerve-fibres and ganglion-cells of her brain, which resulted from the pressure upon it that was exerted by the extravasated blood. The speedy death was due to the circumstance that the medulla oblongata quickly became involved in, and had its functions abolished by the hemorrhage. Thus, the respiratory movements were soon arrested, the circulation of the blood was stopped, and death was produced. This case, then, shows that apoplexy, in the ancient and clinical sense of the term, is sometimes produced by cerebral hemorrhage. It is, nevertheless, true that genuine apoplexy is but seldom due to cerebral hemorrhage, on the one hand, and that cerebral hemorrhage is generally not attended with the phenomena of genuine apoplexy, on the other.

Several things revealed by the autopsy in this case are worthy of special notice. They are, *first*, the remarkable abundance and scope of the extravasation. It occupied the right temporal region, the base of the cerebrum to great extent, the ventricles, and surrounded the pons Varolii and medulla oblongata. The hemorrhage was both meningeal and ventricular, but the former was far more copious and destructive in its consequences than the latter. *Second*, the extravasated blood pressed or encroached upon the pons Varolii and medulla oblongata. This feature of the case confirms, as far as it goes, Trousseau's statement, that "apoplectic stupor is a very exceptional symptom of invasion in cases of cerebral hemorrhage, unless there be lesion of a central part, [of the brain,] or an attack of convulsions." (Vide *Trousseau's Lectures on Clinical Medicine*, vol. i. p. 8, New Sydenham Soc. ed.) *Third*, the brain-substance presented a remarkably exsanguinated appearance, which was doubtless due to compression of its capillaries by the extravasated blood. Now, in cases like the one at present under consideration, the hemorrhage continues until the tension of the effusion equals that of the blood in the ruptured arteries, and as the tension of the blood in the arteries is greater than that of the blood in the capillaries, the last-named vessels yield to or become flattened by the pressure exerted on the brain-substance containing these vessels, by the effusion. The paralysis of the brain and the symptoms of apoplexy which occurred in this case were, in all probability, due to anæmia of the nerve-fibres and ganglion-cells of the brain, produced in the above-mentioned way.

Again, cerebral hemorrhage is not likely to produce that sudden suspension of the cerebral functions which constitutes apoplexy, unless the blood is extravasated suddenly and in great quantity; and for these conditions to be fulfilled it is necessary that the blood should be poured into the arachnoid sac or into all the ventricles, or simultaneously in both situations, as it was in the case we have just related.

The lesion of the kidneys also is worthy of note. At one time it had given rise to anasarca; and it had probably aided in producing a weakened or brittle condition of the cerebral arteries, which rendered them especially liable to rupture. The predisposing cause of the hemorrhage in this case was probably the weakened condition of the cerebral blood-vessels; the exciting cause, the increased tension of the cerebral arteries which was produced by labor and a stooping posture.

CASE XXV.

Puerperal eclampsia and death in less than three quarters of an hour; autopsy; very copious and extensive hemorrhage in the brain, etc.

Mrs. Honora L——, aged 35, married, in respectable but moderate circumstances, pregnant, and near the completion of term, was accidentally injured by falling partly through the deck of a steamboat, while on an excursion in company with her husband. She unwittingly stepped into a hole while walking in the midst of a crowd. This hole was large enough to readily admit one leg, but not her whole body. Her fall was, therefore, suddenly arrested by her thigh striking with great violence against the edge of the hole. It jarred her very much. Subsequently she complained a great deal of pain in the back part of her head. Three weeks after the accident, on Sunday evening, August 8th, she began to have abdominal pains of an intermittent character, in addition to her headache. Supposing her to be in labor, her husband called a physician, who, finding the os uteri unaffected, ordered an anodyne, as follows: ℞. Morphiæ sulph., gr. $\frac{1}{4}$; Pulv. camphoræ, grs. ij.; Pulv. glycyrrhizæ, grs. viij. M. Ft. pulv. S. Take one half of the powder immediately, and the remainder in half an hour, unless sleep has been produced. Directly after taking the first dose she was seized with a fit. She suddenly became unconscious, having convulsive movements of the extremities with convulsive twitchings of the face, and frothed at the mouth. She did not come out of the fit, but died comatose in "less than forty-five minutes." The physician was recalled and bled her freely, but without benefit. The second dose of the anodyne was not administered. Her health had generally been good until the accident occurred.

Autopsy, by the author, nineteen hours after death. Surface of body pale; rigor mortis absent; there were marks of an old bruise on the right thigh, as large as my hand; pupils (both) symmetrically dilated.

Head.—Veins of pia mater contained less than the normal quantity of blood. Substance of brain much exsanguinated. A coagulum, about two ounces in volume, was found in the left lateral ventricle. It was pretty firm in consistence, and reddish-brown in color. The brain-substance surrounding it was more or less broken down and softened to the depth of half an inch. There was no fetor, and we did not find any pus, or other product of inflammation. The right lateral ventricle contained about half an ounce of bloody serum. On the anterior surface of the pons Varolii and medulla oblongata we found a thin layer of blood effused beneath the arachnoid. In the central part of the pons Varolii the nervous tissue was softer than natural, yellowish-white in color, and exhibited blood recently effused into it. This extravasation did not constitute a clot distinctly separate from the softened nervous substance, but was infiltrated through it, thus constituting numerous punctiform effusions. In the central part of the left hemisphere of the cerebellum also a recent coagulum of considerable size was found. It was an inch in length by one third of an inch in diameter, and well rounded off at each end. The brain-substance surrounding it was softened and yellowish-white in color to the depth of something more than one fourth of an inch. No pus or other inflammatory products were detected. The consistence of the brain was natural, excepting the parts of it above mentioned. The softened portions were all either white or yellowish-white in color.

Thorax.—Lungs natural, except that the left was somewhat congested. Heart rather large, and exhibited rather more than the normal quantity of fat on its exterior; valves natural; foramen ovale completely closed.

Abdomen.—Liver and spleen not diseased. Stomach nearly empty. Its mucous membrane was injected with blood in spots along the greater curvature, but otherwise normal. The kidneys were flabby, but in other respects appeared to be sound. The uterine tumor (uterus) contained a healthy-looking fœtus of about the full term. The os uteri was not dilated nor dilatable.

Comments.—This woman's friends thought that she was poisoned by the anodyne powder. But this suspicion had no good foundation, for it was clearly shown at the investigation that the prescription was accurately prepared; and the dose of morphia (gr. $\frac{1}{8}$) was certainly not excessive. Moreover, the fit occurred before the morphia had time to produce much effect, even if a poisonous dose had been administered; and besides the symptoms and post-mortem lesions were not such as are produced by poisoning with opium or morphia.

This was a case of puerperal convulsions or eclampsia. The convulsive movements and the frothing at the mouth, which attended the invasion and progress of her disorder, show conclusively that it was not apoplexy, but a member of the great family of convulsive affections. Her pregnant condition, the near approach of full term, and the occurrence of symptoms which are often connected with the first stage of labor, taken together, make it more than probable that this convulsive disorder was puerperal eclampsia.

When viewed in this light the cerebral hemorrhage which occurred in this case must be considered, not as the primary affection, but as a consequence of the eclampsia. Indeed, it is not difficult to understand how convulsive diseases such as this may powerfully tend to induce cerebral hemorrhage, especially in subjects who are already predisposed to its occurrence, by causing distention of the cerebral blood-vessels.

But it is probable that the injury which she received three weeks before death had also something to do with producing the cerebral hemorrhage. It will be remembered that she was jarred very much by her fall, and that ever afterward she complained a great deal of pain in the back part of her head. We therefore think it not unlikely that her injury entailed such disturbances in the cerebral circulation and nutrition as rendered her more liable to the occurrence of cerebral hemorrhage. The concussion or commotion of the brain, produced by her fall, appears, therefore, to hold the relation of a predisposing cause, and the puerperal eclampsia that of an exciting cause of the extravasations in her brain. The apoplectic stupor or coma which attended the invasion was produced, not by the hemorrhage, but by the convulsions or eclampsia.

Did the softening of the brain-substance around the clots which constituted a marked feature at the autopsy precede the hemorrhage? With Trousseau, the author holds that it did not. That eminent authority says: "Agreeing in this with the majority of medical men, I believe that the softening of the brain which accompanies hemorrhage is an effect, and not a cause." (*Op. cit.* vol. i. p. 9.) The autopsy in this case did not furnish any evidence that it was due to a preëxisting encephalitis. Moreover, the symptoms would not warrant the belief that she had suffered from inflammation of the brain for three weeks before death. The cerebral softening in this case was doubtless a purely mechanical effect of the extravasations.

Case XXVI.

Epileptiform convulsions occurring in an intemperate subject; the second paroxysm terminated in death; autopsy; brain atrophied, indurated, and congested; meninges congested and œdematous; ventricular hemorrhage; also very extensive meningeal hemorrhage.

John M——, aged 46, had been a hard drinker of spirituous liquors, especially brandy, for sixteen years. On the 10th, 11th, 12th, and 13th days of November he was absent from home on a debauch. He returned on the last-mentioned date, and said he felt very sick. In the evening of the same day he complained of headache, vomited frequently, and sweat profusely. He vomited also on the next day, Sunday, Nov. 14th, and in the evening was seized with an epileptic fit. He lay in deep stupor and had stertorous breathing for some time after the convulsive movements ceased, but finally recovered consciousness. On the following day, Monday, Nov. 15th, he had another attack of epileptiform convulsions, and died rather unexpectedly about 3 o'clock P.M., comatose. No physician was called.

Autopsy, by the author, twenty hours after death. The corpse was not fat, and exhibited a yellowish or semi-icterode hue. The post-mortem rigidity was but moderate.

Head.—The glandulæ Pacchioni were very large, and formed pretty strong adhesions between the arachnoid and dura mater at the vertex. The visceral arachnoid exhibited pearly opacity, without thickening, in spots and streaks, particularly over the track of the large blood-vessels of the pia mater. There was also an abundant effusion of pale serum in the subarachnoid connective tissue, especially at the vertex, which gave it a gelatinous appearance in that situation. The vessels of the pia mater and substance of the brain were strongly congested. We also found a large quantity of coagulated blood occupying the following situations, namely, the fissura Sylvii of both hemispheres, the track of the optic nerves, the exterior of the pons Varolii and medulla oblongata, the base of the cerebellum, and the fourth ventricle. We did not find the vessels from which this hemorrhage had sprung. The substance of the brain had a good color and a very firm consistence, that is, it was indurated. The blood-vessels belonging to it exhibited well-marked dilatation, produced probably by chronic congestion. The lateral ventricles contained a small quantity of limpid serum. But little fluid of any kind escaped from the skull during the whole examination.

Thorax.—The lungs contained more than the normal quantity of blood, but in other respects were natural. The heart was rather large and flabby. Its muscular structure was undergoing fatty degeneration, and had a dingy, brownish-yellow color, resembling that of decayed leaves. The walls of the left ventricle were so much softened that I thrust my finger through them with ease. The valves were natural.

Abdomen.—The liver was large, its edges were blunt but not rounded off, its surface was smooth, and had a uniform pale-yellow or buff color. It presented the same color as a liver taken from a patient who died of yellow fever, which I had seen a few days before. Its texture was not softened. The spleen was normal in size but rather soft in consistence. The kidneys were very large, pale, mottled, and contained more than the normal quantity of blood. On making an incision through the cortical substance, the cut surface was found to present a pale-yellow color, liberally specked with little pale-red spots of an irregular shape. The substance of both organs was quite friable. The stomach was capacious, congested, and contained a few ounces of liquid having a brownish color. Its mucous membrane was mammillated and pale in color in the pyloric portion of the organ, but it was dark-colored at the fundus. The gastric mucous membrane was also softer than natural throughout its whole extent. The intestines were congested.

Comments.—That the brain in this case was atrophied was proved by the dilated and congested condition of the blood-vessels which permeate its substance, by the strongly marked œdema and congestion of its membranes, and by the copious extravasation of blood; for these several lesions could not have presented themselves coincidently, or met together in the same case, unless the brain was much shrunken or diminished in size, as we have already abundantly shown.

This man had epilepsy induced by intemperance, and died in a paroxysm of that disorder. The immediate cause of death was a cerebral hemorrhage, which paralyzed the medulla oblongata. The extravasation of blood, however, was an accident which attended the epileptic seizure, and is an occurrence which at any time is liable to be produced by epileptic convulsions, especially in intemperate subjects. It is plain, then, that this man was stricken down, not by apoplexy, but by epilepsy, and that the disorder which destroyed him was in reality epilepsy, and not apoplexy. Cases like the one just related are not unfrequently described as apoplectic strokes. Such a view of these cases, however, is erroneous, for in such cases the apoplectic stupor or coma is produced by the epileptic seizure itself, and not by the hemorrhage. They are truly attacks of epilepsy which have afterward become complicated with cerebral hemorrhage, and should be regarded only in that light. When patients survive epileptic seizures, the risk to which they are more especially exposed is a return of the epilepsy, and not the occurrence of cerebral hemorrhage. The essentially convulsive nature of such attacks is important in a clinical point of view, and should not be overlooked.

Thus we find that the relationship between epilepsy and apoplectiform disorders is very intimate, and one which we are sometimes liable to overlook unless our attention is constantly directed to it. On this topic Dr. Copland very justly observes: "The connections of *apoplexy* and *palsy* with *epilepsy* are more intimate than has usually been remarked, or even admitted by modern writers, who have generally been more prone to point out distinctions or to establish differences, than to record intimate alliances, or even still more close connections, not only between the diseases just named, but also between others similarly circumstanced. These maladies, although not similar in many respects, are nevertheless so intimately related, as respects frequency of succession, and the nature of the organic changes of which they are the outward manifestations during life, as to require some notice of their connections at this place: 1st, An attack, more or less sudden, may present the mixed characters of apoplexy and epilepsy. 2d, The epileptic seizure may pass into the apoplectic. 3d, The epileptic attack may be followed by paralysis, either directly or as a consequence of either of the two preceding forms of seizure; and 4th, The paralytic affection may be followed, although in rarer instances, by an epileptic attack, or by coma attended by convulsions, most frequently terminating in death." (Vide *Copland on Palsy and Apoplexy*, pp. 173, 174.)

This case differs considerably from the two preceding instances of cerebral hemorrhage which we have related, in respect to the amount of blood that the vessels of the brain and its membranes were found to contain, when examined after death. In this case the brain and its membranes were strongly congested. In the other two cases they were not congested, but, on the contrary, they were decidedly exsanguinated. Nor is it difficult to account satisfactorily for this important difference. The shrunken brain and intemperate habits of the epileptic subject, aided, in all probability, by the powerful character of the convulsive movements, had caused his head to become much congested, and the cerebral blood-vessels to become greatly distended, so that finally, under the influence of this internal pressure, some of them burst open, and blood in great quantity was extravasated upon and within the brain. These vessels burst not so much because their walls were weak, as because they were over-distended with blood, while in the other two cases rupture and consequent hemorrhage occurred mainly because the strength of the vascular tunics had become impaired by local disease.

The copious effusion of serum which was found beneath the visceral arachnoid membrane in the case last related, resulted, first, from cerebral congestion, and second, from some depraved condition of the blood itself which favored the separation of the amorphous elements from it.

The substance of the brain in this case was decidedly firmer in consistence than natural, or indurated throughout its whole extent. The extravasated blood was abundant in quantity and widely diffused. This case, then, proves Rouchoux's idea, that cerebral hemorrhage is necessarily preceded by cerebral softening, to be incorrect. He held that softening of the cerebral substance always precedes cerebral hemorrhage; that the former paves the way for the latter, and accordingly he termed it "hæmorrhagipare;" but he was mistaken.

The induration of the brain-substance in this case was due to alcoholic intemperance. We have already related with more or less fulness a considerable number of cases in which chronic alcoholism and its lesions constituted an important feature. It may be both interesting and useful to sum up these lesions in this place. Our autopsies show that alcoholic intemperance when long continued generally produces the following lesions of the cerebral substance, namely, *atrophy*, *induration*, and *dilatation of the cerebral blood-vessels*. These lesions are more or less fully developed in almost all old spirit-drinkers. But atrophy of the brain may also be induced by other causes, such, for example, as old age, exhausting disease, etc. In such cases, however, it is very often accompanied by more or less softening of the brain. Again, dilatation of the cerebral blood-vessels may be produced by any other form of chronic cerebral congestion, such, for instance, as results from long-continued mental excitement, the abuse of narcotics, etc. But induration of the brain-substance is not produced by any other cause with any thing like the same frequency, and to any thing like the same extent, as it is by chronic alcoholism. Cerebral induration is, therefore, the lesion which characterizes the brains of drunkards more than any other. Occasionally, however, the cerebral substance is found to be softened in old topers, (see Case VI.,) but this is quite exceptional. The other lesions which may be induced by chronic alcoholism are hypertrophy of the heart, fatty degeneration of the liver, kidneys, and heart, and thickening with softening of the gastric mucous membrane, according to the author's experience.

CASE XXVII.

Sudden death from cerebral hemorrhage; phenomena attending it unknown; autopsy; hemorrhage both meningeal and ventricular; substance of brain exsanguinated.

A man, aged about 35, was found lying dead in the street, by a police officer, on the evening of September 4th. It was supposed that he had been killed by a blow on the head. It appeared that he was a hard-working laborer, and sometimes addicted to excessive drinking; that on the evening of his decease he was much the worse for liquor, but not absolutely drunk, and that he had a warm dispute, becoming very angry, with an acquaintance in a porter-house a little while before he was found dead.

Autopsy, by the author, fifteen hours after death. The whole surface of his body was pale, except the face, which was rather bloody from nasal hemorrhage, occasioned, perhaps, by bruising his nose in falling. The pupils were natural.

Head.—There was neither ecchymosis nor any other mark of violence on the scalp. The skull was thicker than natural. We found a thin layer of coagulated blood effused into the connective tissue beneath the visceral arachnoid, so as to fill the sulci and overspread the convolutions, throughout both temporal regions, and likewise at the base of the brain. The coagulum was most abundant in quantity about the corpora quadrigemina, the medulla oblongata, and the base of the cerebellum. There was also a good-sized clot of blood (about as large as a peanut with the shell on) in the anterior part of each lateral ventricle, the left clot being a little larger than the right one. While examining the brain, four or five ounces of bloody serum flowed away. We did not find the ruptured vessels from which the hemorrhage occurred. The substance of the brain contained less than the normal quantity of blood, that is, it was exsanguinated, but in other respects seemed to be natural.

Thorax.—The lungs contained more than the normal quantity of blood. The heart was a little larger than it should be, and its muscular tissue was pale and flabby.

Abdomen.—On opening the stomach the odor of spirits became ap parent, and the gastric mucous membrane was seen to be congested in spots. The liver exhibited fatty degeneration in an early stage.

Comments.—The hemorrhage in this case was principally meningeal. The clots in the lateral ventricles, however, were so large that they would have been considered important if there had been no meningeal extravasation. But the most important seat of the hemorrhage was at the base of the brain. There the blood gathered in such quantity as to compress the medulla oblongata, and cause anæmia of its nerve-fibres and ganglion-cells, thus suspending their functions and arresting the respiratory movements.

The absence of all signs of contusion from the scalp proves that this man was not killed by being struck on the head. Now, the question arises, whether the cerebral hemorrhage in this case occurred spontaneously or resulted from an attack of epilepsy, or eclampsia, or from falling by accident in the street. In the absence of witnesses, the solution of this question must be left to conjecture; and, taking all the circumstances into consideration, we are inclined to adopt the last-mentioned hypothesis. That he was much the worse for liquor, and in a state of great mental excitement on the fatal evening, we have undoubted proof. That he fell on his face in the street the nasal hemorrhage renders probable. Now, we think it much more likely that he fell down upon the pavement from intoxication, than that he was struck down by disease. Moreover, he was still young, his age being only about 35; he had always been healthy, and the autopsy showed that he was not yet affected, to any considerable extent, with the lesions which characterize alcoholismus chronicus. The concussion of the brain which would result from falling down in the street might readily suffice to cause some of the congested vessels of the meninges and plexus choroideus to burst, and extravasation of blood to occur. We therefore hold the opinion that the cerebral hemorrhage in this case was directly induced by injury in the nature of concussion of the brain, and that the predisposing cause was intense cerebral congestion, due to mental excitement and to alcoholic drink.

Formerly it was supposed that meningeal hemorrhage was generally, if not always, due to injury. But this opinion is not correct, for meningeal hemorrhage not unfrequently occurs spontaneously, as well as in consequence of the operation of violent causes. Of the four cases in which more or less meningeal hemorrhage occurred, that we have thus far related, the extravasation appears to have been directly due to injury in but one nstance.

Case XXVIII.

Sudden death from coma after a beating; autopsy; extensive meningeal hemorrhage; kidneys strongly congested, etc.

July 27.—The author made a post-mortem examination, at the Fourteenth Ward station-house, of the body of Perry Brinsley, aged 30. Deceased was said to have been beaten by somebody in Elizabeth street, on the 26th. He was, however, able to get away without assistance, but soon afterward was found lying unconscious and in a dying condition. He had drunk hard for some considerable time previously. The beating, after all, may have been apocryphal and imaginary, as no arrest was made, and no witness of it was produced.

Autopsy at 11 A.M., about eighteen hours after death. On inspecting the cadaver, we found a bruise on the right cheek, just below the eye, and some dark-colored tumefaction of the jaw and neck in the neighborhood of the right ear; but these parts were so much affected by post-mortem changes as to prevent deciding whether this discoloration was connected with contusion or not. Found also a slight bruise on the top of the right shoulder, and another on the left arm near the elbow.

Head.—On dissecting off the scalp, no evidence of contusion was brought to light. The skull was not fractured nor injured in any respect. On removing the skull-cap and turning back the dura mater, the convex surface of both cerebral hemispheres was found to present a dark-brown color, from extravasated blood in the connective tissue beneath the visceral arachnoid. This effusion was particularly abundant in each temporal region, but rather more so on the right than on the left side; also at the base of the cerebrum, about the origin of the optic nerves, on the anterior aspect of the medulla oblongata, and at the base of the cerebellum. The sulci were filled with coagulated blood over the whole convex surface of the cerebrum. But little fluid of any kind escaped on removing the calvaria. The ventricles contained about two dessert-spoonfuls of bloody serum. A clot of blood as large as a peanut was found in each lateral ventricle, that belonging to the left being in the anterior horn, and that belonging to the right in the posterior horn. The substance of the brain was congested throughout, but its consistence was normal.

Thorax.—The lungs exhibited post-mortem congestion or suggilation. The heart was rather fatty, and contained no clots. The foramen ovale was completely closed.

Abdomen.—The liver and spleen were hypertrophied. The veins of the stomach were large and full of blood, but the organ itself was nearly empty. Its mucous membrane was colored reddish-brown in spots upon both curvatures, and near the cardiac orifice. This mucous membrane was also somewhat thickened and softened generally. The kidneys were large, dark-colored, and intensely congested. On section, their substance presented a nearly uniform reddish-brown or maroon hue. Their consistence was firm and good.

Comments.—What caused the meningeal hemorrhage in this young man's case? He was not supposed to have been much injured until he was found in a dying condition. It appears that he was not struck senseless by the blow or blows which he was supposed to have received. It seems that he was able to walk, and did not lose consciousness until some little time afterward. Thus it is proved that he was not bereft of sense by the shock of blows. Still, the meningeal hemorrhage may have been due to injury; for, if the blood was slowly extravasated from the ruptured vessels, some time would probably elapse before the quantity would become sufficient to abolish consciousness and produce coma. It seems, however, more likely that he was seized with uræmic eclampsia or coma, and that this disorder produced the apoplectic stupor and the meningeal hemorrhage. This view of his case is considerably strengthened by the fact that his kidneys were much diseased, being enlarged, discolored, and strongly congested. Indeed, their morbid state was substantially the same as that which has been found in undoubted cases of uræmic eclampsia.[1]

The evidence afforded by the autopsy in this case is not decisive as to whether the meningeal hemorrhage was the result of injury or not. The weight of it, however, inclines to the latter view, for the scalp and skull were found entirely free from injury, while the kidneys were seriously diseased; and renal disorder *per se* strongly predisposes to the spontaneous occurrence of cerebral hemorrhage.

The brain in this case was beginning to show the consequences of chronic over-indulgence in spirituous drink. Its substance was congested, and considerable atrophy of it must already have taken place; otherwise, the hyperæmia could not have been associated with the large amount of extravasation which was present.

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[1] Wilks, in a paper on meningeal hemorrhage, both spontaneous and traumatic, calls attention to the occurrence of dubious cases, in which, except disease of the blood-vessels, the autopsy reveals nothing to aid in determining whether the hemorrhage was spontaneous or not. (Vide *New Sydenham Soc. Year-Book*, 1859, pp. 195, 196, and *Guy's Hospital Reports*, 1859, pp. 281–304.) But an inquiry into the condition of the kidneys, in respect to disease, and of the post-mortem urine in respect to containing albumen, may also afford useful information. It may serve to show that convulsive movements which existed during life were due to uræmic eclampsia, and perhaps, that the hemorrhage itself was induced by the convulsive disorder.

CASE XXIX.

Sudden death, attended probably by epileptiform convulsions; autopsy; profuse meningeal hemorrhage; substance of brain exsanguinated; liver much enlarged and fatty.

Mrs. Matilda S——, age 21, married, and the mother of two children, was found lying dead in her apartments on the morning of May 3d. Her husband was absent at his work. It appears that they had moved into new apartments on the evening of Saturday, May 1st, and that they had celebrated the event by hard drinking, continued through Sunday, the 2d. Early on the morning of the 3d, her husband went to his work, as usual, leaving the deceased at home, apparently well, where she was found dead by the neighbors, a little while afterward.

Autopsy, by the author, about twelve hours after death. We found the body lying on a pallet, on the floor; decubitus partly on the right side and back. Bloody, frothy fluid was issuing from the mouth and nose. The body was dressed in ordinary clothing, and was that of a rather small and delicate woman. It was well preserved, and the rigor mortis was strong. There was a slight bruise on the top of the right shoulder, and another on the left arm. The cadaveric discoloration was abundant on the right arm, on the right side of neck, and on all the depending portion of her body. Observed that she had passed urine freely where she lay.

Head.—There was no lesion of the scalp or skull. After removing the calvaria and raising the dura mater, we found more than four ounces of fluid and coagulated blood lying in the cavity of the arachnoid, that is, on its free surface, spread out over the exterior of the *right* cerebral hemisphere. The coagulum alone, when gathered up, was fully equal to three ounces in volume. We failed to discover the source of the hemorrhage. The lateral ventricles contained a small quantity of pale serum. There was also some bloody serum about the right hemisphere of the cerebellum. The brain generally contained less than the normal quantity of blood. Its substance was paler than natural, the puncta vasculosa few and small. Its consistence was normal throughout.

Thorax.—The lungs contained more than the normal quantity of blood, and the air-passages were filled with bloody, frothy fluid, but presented no other lesion. The heart was natural, except the mitral and aortic valves, which were slightly thickened; they did not seem to be so much thickened, however, as to occasion any mischief.

Abdomen.—The liver was much larger than natural, and fatty. It had a pale yellow or straw color, a soft consistence, an oily feel, and greased the knife on cutting into it. The spleen was rather above the normal size, and somewhat softened. The stomach was filled with gases, but empty of liquids and solids. Its mucous membrane was deeply injected with blood at the fundus and along the greater curvature, and it was more or less injected throughout the rest of its extent. The kidneys were fatty. The intestines appeared to be natural.

Comments.—It is probable that this woman felt badly, and laid down again after her husband's departure to his work. The previous debauch and the undisturbed state of her clothing seem to warrant this opinion. It is also probable that she had an attack of epileptiform convulsions as a sequel to her debauch, for her body, when found soon after death, still exhibited frothing at the mouth. Moreover, she passed urine involuntarily during the fit, as those having epileptiform convulsions are apt to do. She suddenly lost her consciousness, in all probability, or she would have called for assistance. Granting the presence of these three symptoms, sudden loss of consciousness, frothing at the mouth, and involuntary discharge of urine, we may be sure that the subject also had convulsive movements of an epileptic or eclampsic character, for no disease besides epilepsy and eclampsia is attended by this combination of phenomena. It is, therefore, almost certain that this woman had an attack of epileptiform convulsions, and that this disorder induced apoplectic stupor, meningeal hemorrhage, coma, carus, and death, in a brief space of time. Here, then, we have another instance in which well-marked meningeal hemorrhage was not due to injury. We must consider that her case belongs to the same category as Cases XXV. and XXVI., both of which had epileptiform convulsions, and that the meningeal hemorrhage occurred as a complication of the epileptiform seizure, although it proved to be the proximate cause of death.

CASE XXX.

Eclampsia occurring in a new-born child, and death ; autopsy ; extensive meningeal hemorrhage, the result, in part at least, of injury.

On the night of Thursday, August 5th, about eleven o'clock, a fresh-born female infant was found exposed and naked, in the vault of a privy belonging to a house in Mercer street, where it had been thrown for the purpose of concealing its birth. Attention was attracted to the little being by its cries. In a few minutes it was removed from its perilous situation. A physician saw it, and finding the umbilical cord not tied, applied a ligature. The cord did not bleed. Its free extremity looked jagged, as if it had been torn asunder. On making search, its brutal mother was found, and the next morning she, with the infant, was sent to the City Prison. During the day (Friday) the child appeared to be "pretty smart," according to the statement of the physician of the prison. But, on the next morning, (Saturday,) it was seized with convulsions. The doctor noticed some bruises on its head. About two o'clock P.M., it died in a convulsion.

Autopsy, by the author, twenty-two hours after death. Cadaver presented the appearance and odor of incipient decomposition. The cuticle was loosened on the breast. The umbilical cord was exsiccated and contained in the proper dressing. The scalp exhibited the marks of a bruise over the frontal bone, on the left side, near the coronal suture. The scalp was bruised in two other places in the left parieto-occipital region. On removing the calvaria and dura mater, the surface of the middle and posterior lobes of the left hemisphere of the cerebrum was found to be smeared over with extravasated blood. The sulci of the convolutions were also filled with it in the same locality. Some extravasated blood was found at the base of the cerebellum on each side, together with an abundant quantity of bloody serum. The substance of the cerebellum appeared to be somewhat softened. No other evidence of inflammatory action was found. The skull was not fractured.

Each pleural cavity contained a small quantity of bloody serum. The lungs were well expanded, crepitant, etc. The upper lobe of the right lung was less crepitant than the other portions. The ductus arteriosus presented a shrunken and wrinkled appearance. Its calibre had undergone considerable diminution.

The stomach was contracted and nearly empty. The intestines were empty of meconium, and presented a pale color externally.

Comments.—The extravasation of blood upon the brain, or meningeal hemorrhage, which was found in this case, was probably occasioned by the injury the head had sustained. Some of the vessels of the pia mater on the left hemisphere were lacerated by the same force which contused the scalp. At first the extravasation was small in quantity, and amounted to but little more than ecchymosis. When convulsions occurred, the vessels of the brain and its membranes became congested, and the hemorrhage returned in great quantity. (Vide *New-York Journal of Medicine, August*, 1867, pp. 393–395.) When cerebral hemorrhage occurs during infancy or childhood, the blood is generally effused into the cavity of the arachnoid, and into the subarachnoid connective tissue, as it was in the case just related. Hemorrhage, however, does sometimes occur in the substance of the brain even during infancy, but this form of extravasation is rather uncommon during the first period of life. This subject will be more fully discussed in the chapter on infantile apoplexy. (See Chapter VIII.)

We have thus far in this chapter related only such cases as properly came under the head of apoplectiform cerebral hemorrhage, that is, cases in which the invasion was characterized by that sudden loss of consciousness which under another name is known as apoplectic stupor, or, in other words, cases in which coma was present from the very outset of the attack. Those other and more common cases of cerebral hemorrhage in which the intelligence is not much affected at the beginning of the attack, or the consciousness is only momentally or incompletely destroyed, and is soon recovered again whatever may subsequently happen, cases which are sometimes classified under the head of paralysis, (hemiplegia,) do not belong to the category of apoplectiform cerebral hemorrhage, and are, therefore, strictly speaking, foreign to our theme. We shall, however, give some considerable account of these cases, in order to get a more clear understanding and a more comprehensive view of the subject of cerebral hemorrhage. But, before doing so, we shall briefly sum up the seven cases which we have just related.

In but one of these seven cases (Case XXIV.) was the sudden loss of consciousness or apoplectic stupor, which attended the invasion, induced by the extravasation itself. In six of them it was due to either epileptiform convulsions or eclampsia, (Cases XXV., XXVI., and probably Cases XXVIII., XXIX., and XXX. also,) or to the shock of injury, (Case XXVII. probably.) With regard to the effect of both epileptic and eclampsic convulsions on the consciousness, Trousseau has established the following proposition: "The same cerebro-spinal modification which causes the fit of epilepsy or eclampsia, the insultus, the ictus epilepticus, is sufficient to produce the apoplectic stupor which follows it." (Vide *Lectures on Clinical Medicine*, vol. i. p. 36.) In the epileptiform and eclampsic cases the cerebral hemorrhage occurred as a consequence and complication of the convulsive disease. But one of these seven cases, therefore, was in reality an instance of apoplexy. This circumstance goes to show how infrequently hemorrhagic apoplexy, in the correct sense of the term, is met with. Thus our experience supports the views of Trousseau, which we have already pretty fully stated, on this important point.

In six cases the extravasation was principally meningeal in character. (Cases XXIV., XXVI., XXVII., XXVIII., XXIX., and XXX.) The blood was effused into either the cavity of the arachnoid, or into the subarachnoid connective tissue, or simultaneously in both situations; and in the first four instances mentioned above, some blood found its way into the ventricles of the brain also. In all these cases of meningeal hemorrhage the extravasation was copious and extensively distributed; and in the four instances just mentioned it occasioned death by directly compressing the medulla oblongata. In none of these six cases of meningeal hemorrhage was there any laceration of the cerebral substance or any cerebral aneurism, and in each instance we failed to discover the source of the extravasation. In but one instance (Case XXV.) was the hemorrhage not principally meningeal in character. In it the blood was mainly extravasated into the left corpus striatum, from which, however, it burst into the left lateral ventricle; also into the substance of the pons Varolii, and into the substance of the left hemisphere of the cerebellum. The ruptured vessels in this case were not discovered.

These victims of cerebral hemorrhage were mostly remarkably young. One was a young infant, another was aged only 21, another 23, another 30, two were 35, and only one as old as 49. We may further remark concerning the etiology of the hemorrhage in these cases, that in five of the six adults the kidneys were considerably diseased, and it is believed that these organs were unsound in the remaining adult, although the record is silent on this point, for he was an intemperate subject. This remarkable relationship between chronic disease of the kidneys and cerebral hemorrhage is probably something more than a mere coincidence. Rokitansky and Virchow have both described a peculiar state of the walls of the minute blood-vessels, in which, without any histological alteration of structure, they become weakened, and their elasticity is impaired, through imperfect nutrition. They consider chronic disease of the kidneys as the chief cause of this peculiar change, adopting in this respect the opinions of Bright, Burrows, Gregory, Christison, Kirkes, and Rayer. It is probable that such a state of the minute blood-vessels of the brain and its membranes was present in at least some of the cases which we have related, and assisted in producing the cerebral hemorrhage. But the further discussion of this point must be postponed until the summing up of our cases is completed.

Some of the views which are generally entertained concerning meningeal hemorrhage are probably erroneous. For example, Niemeyer says, "Excepting traumatic hemorrhages of the meninges, among which are to be classed those occurring during birth, this is a rare affection." (Vide *Text-Book of Practical Medicine*, vol. ii. p. 202, Am. ed.) Now, of our six cases of meningeal hemorrhage, three were certainly not due to injury, another probably belonged to the same category, leaving only two cases in which it was probably due to traumatic causes. According to our experience, therefore, meningeal hemorrhage of spontaneous origin is not a rare affection. Again, Niemeyer says, "Effusions of blood in the subarachnoid space or between the dura mater and arachnoid result mostly from the breaking through of a cerebral hemorrhage." (Vide *op. cit.* vol. ii. p. 202.) This did not occur in any of our cases, and we therefore think that this statement also is erroneous.

The cases which we have related show that meningeal hemorrhage is apt to prove quickly fatal. This is doubtless due in part to the great rapidity with which the blood can be effused, and the great facility with which the medulla oblongata can be compressed, when it occurs.

Our cases also show that cerebral hemorrhage generally produces anæmia of the cerebral substance, or, in other words, that compression of the brain from hemorrhage generally occasions compression of the cerebral capillaries. In four cases we found that the substance of the brain presented an exsanguinated appearance at the autopsy, in two a congested appearance, and in one the condition is not stated, but in all probability it was anæmic. The two subjects in which the brain was found to present a congested appearance after death, notwithstanding the extravasation of a large quantity of blood upon it, were both very intemperate, and this circumstance probably explains the apparent anomaly. In one of them the brain was much atrophied, its substance indurated, and its blood-vessels dilated. In the other there was cerebral atrophy, but no induration was observed. It therefore appears that when cerebral atrophy is present, cerebral hemorrhage is less likely to produce anæmia of the brain-substance than it is when the brain is normal in size. The enlargement in the calibre of the cerebral blood-vessels, which intemperance occasions, also assists in explaining the congested appearance which these two brains presented.

Trousseau has shown that when hemorrhage occurs in a central part of the brain, such as the pons Varolii, a part in which all the nerve-fibres going from or coming to the cerebrum come together and decussate, it may produce the phenomena of apoplexy properly so called. In our own case of hemorrhagic apoplexy, (Case XXIV.,) however, the extravasation occurred into the cavity of the arachnoid; and in the only undoubted case of apoplexy related by Mushet the hemorrhage was also meningeal. (Vide *Mushet on Apoplexy*, p. 35.) But, in order for cerebral hemorrhage to be attended with the phenomena of apoplexy, it is, for the most part, necessary that the effusion should be very sudden or rapid in occurrence, and abundant in quantity. For when the effusion occurs slowly, or is small in quantity, the earlier phenomena are always those of paralysis, in the form of hemiplegia, and more or less complete. When the substance of the cerebrum is the seat of hemorrhage, apoplectic stupor generally does not attend the invasion, because in such cases the extravasation usually occurs much too slowly to produce this symptom. On examining post mortem the cases in which hemorrhagic apoplexy, properly so-called, has occurred, we shall generally find that the blood has been effused into the cavity of the arachnoid, (that is, on the free surface of the brain,) or into the ventricles, or simultaneously in both of these situations. Here the extravasation can be poured out with sufficient rapidity, and in sufficient quantity, to suddenly suspend the functions of the cerebrum, and induce the symptoms of apoplexy, in the ancient and clinical meaning of the term.

Etiology of Cerebral Hemorrhages in general.—Cerebral hemorrhage almost always takes place from the smaller arteries and capillaries of the brain. Its occurrence is promoted by certain diseases of the walls of the cerebral arteries, by increased pressure of the blood against the walls of these vessels, and by certain disordered states of the blood itself.

Charcot and Bouchard, examining the causes to which cerebral hemorrhages are usually referred, find that they may be arranged in three groups: (1) Diminution of the consistence of cerebral tissue to such a degree that it does not furnish sufficient support to the vessels; (2) Increased tension of the blood, depending on hypertrophy of the left ventricle, atrophy of the kidneys, etc.; (3) Diminished resistance of the cerebral vessels in consequence of morbid change in the walls, (fatty degeneration or atheromatous incrustation.) Some of these (as cerebral softening, for example) are doubtful; others appear to be only accessory. Thus the authors find, with regard to arterial atheroma, that it is not present in twenty-two per cent of cases, and that it is only present in a marked degree in twenty-five per cent. Again, hypertrophy of the heart was not present in forty per cent of cases. These statistics are founded on the examination of sixty-nine cases. The only pathological condition which MM. Charcot and Bouchard have constantly met is an aneurismal state of a certain number of the small intra-cerebral vessels. From the small size of these aneurisms the authors give them the name of miliary aneurisms. They are visible to the naked eye. They appear as small globular grains, varying in diameter from two tenths of a millimetre to a millimetre, and sometimes a little more than that. If they contain liquid blood, their color is red or violet; if, on the other hand, the blood is coagulated or has undergone transformation, the aneurism is red-brown, ochry, or even blackish. The color is also influenced by the variable thickness of the wall. The optic thalami, the corpora striata, the convolutions, the pons, the cerebellum, the centrum ovale, the middle peduncles of the cerebellum, the cerebral peduncles, the medulla oblongata are the parts in order of frequency in which these aneurismal dilatations have been met by the authors.

This distribution, they remark, agrees with that of cerebral hemorrhages. Seen under the microscope with a low power, the vessels present a fusiform or sacciform dilatation. With a higher power, it is seen that the wall of the aneurism is continuous with the tunics of the vessel which is its seat; but the three tunics are not distinct; they are fused, and the thickness of the aneurismal wall is less than that of the three normal coats of the vessel, a fact which accounts for their proneness to rupture. Careful observation of the aneurismal vessel reveals alteration of structure, (*arterite sclĕreuse.*) This change proceeds from without inward, for the most considerable changes have their seat in the outer part of the vessel, and the atrophy of the muscular coat depends on change in the adventitious tunic, whence the name, periarteritis, which they propose for it. (Vide *Half-Yearly Abstract of Med. Sciences*, vol. li. pp. 3, 4.)

Professor Béhier confirms the pathological discovery of Charcot and Bouchard, that small aneurisms of the capillary arteries of the brain are generally present in cases of cerebral hemorrhage. These are not like the aneurisms which occur upon a large trunk; they are changes in the minute vessels which commence with a sclerosis of the general arterial system of the brain. Small, true, aneurismal, bladder-like dilatations make their appearance, sometimes only bulging out on the side, but more frequently presenting fusiform enlargements. To the naked eye, as seen on the surface or in the substance of the brain, they appear as dark-red or blackish objects hardly the size of a millet-seed. They are found in the centre of the hemorrhagic effusion itself, and communicate with it by an opening which in all respects resembles the rupture of a common aneurism. Around the rupture one finds the remnants of the membrana adventitia. Occasionally the blood collects between the middle coat and the surrounding adventitia, but finally bursts through the latter. In such cases the coagula contained in the aneurismal cavities are continuous with the blood-clots which form the cerebral effusion. Prof. Béhier also declares: "Since the moment when my attention was first called to this subject, I have discovered similar aneurisms in all the cases of cerebral hemorrhage in which I have been able to make the post-mortem examination. This lesion, therefore, may be viewed as one of the principal causes of cerebral hemorrhage. I do not pretend to say that it exists in every case, but I fully believe that, on proper investigation, it will be found in the great majority of cases." (Vide *Med. Times and Gazette*, Nov. 16th, 1867, pp. 533, 534, and *New Sydenham Soc. Retrospect*, 1867–8, p. 87.)

Another lesion, which some very eminent authorities have thought to be connected with the production of cerebral hemorrhage, is fatty or granular degeneration of the capillaries of the brain. This morbid process, which Robin discovered in 1849, was carefully investigated by Paget in 1850; he has accurately described the formation of these small fatty deposits, which gradually coalesce into larger masses, and thus seriously diminish the resisting power of the small arteries of the brain. This fatty or granular degeneration of the walls of the cerebral capillaries is probably an occasional cause of cerebral hemorrhage.[1]

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[1] We have already mentioned a peculiar morbid state of the walls of minute vessels, in which, without any histological alteration of structure, their elasticity is impaired, that Rokitansky and Virchow have both described. They consider disease of the kidney as the principal cause of this peculiar change. It probably has something to do with the production of cerebral hemorrhage, in at least occasional instances.

The atheromatous condition of arteries, which was mentioned first by Abercrombie as connected with cerebral hemorrhage, has been taken up by later writers. Bouillaud, Grisolle, Rokitansky, Valleix, Niemeyer, Eulenberg, and others consider it as the chief cause of this hemorrhage. Professor Béhier, however, does not coincide in this opinion, but at the same time he admits atheroma to be the cause of meningeal hemorrhage in a considerable proportion of cases. He also says: "One of the very first autopsies which I made in this hospital exhibited a rupture of the middle meningeal artery, which was exceedingly atheromatous. But it must be acknowledged that in cases of encephalic apoplexy the arteries of the brain are generally free from similar deposits." Atheroma was not observed in any of the cases of cerebral hemorrhage which we have related.

Intra-cranial aneurisms, in the ordinary sense of the term, by bursting, sometimes produce cerebral hemorrhage. Indeed, it is not impossible that hemorrhagic apoplexy is due to cerebral aneurism much oftener than has usually been supposed, for Gull has shown that this variety of aneurism is much less rare than has generally been believed. This writer has collected sixty-two cases of intra-cranial aneurism, involving the following arteries, namely, the vertebral in four instances; the basilar in twenty; a small artery in substance of pons in one; the posterior cerebral in three; the internal carotid by sella turcica in eight; the middle cerebral in fifteen; the anterior cerebral in six; the anterior communicating in one; the posterior communicating in four; total, sixty-two. (Vide *New Sydenham Soc. Year-Book*, 1859, p. 196; and *Guy's Hospital Reports*, 1859.) Dr. Hutchinson, of Philadelphia, however, has made a still larger collection, for he has tabulated eighty-five cases of intra-cranial aneurism, in which the following arteries were the seat of lesion, namely, the basilar in twenty-five instances; the middle cerebral in twenty-six; the internal carotid in ten; the anterior cerebral in nine; the anterior communicating in five; the vertebral in four; the posterior cerebral in two; the posterior communicating in two; the superior cerebellar in one; and the middle meningeal in one; total, eighty-five. (Vide *Pennsylvania Hospital Reports*, vol. ii. 1869.)

Increased tension of the blood in the cerebral arteries is produced by hypertrophy of the left ventricle of the heart, by disease of the kidneys, by the plethoric or distended state of the vascular system in general, which results from over-indulgence in food and drink; also by great mental activity, and by straining, more especially by straining at stool. Hence cerebral hemorrhage not unfrequently attacks high-livers soon after indulging in a hearty meal with wine; or public speakers, such as clergymen and advocates, while engaged in pulpit or forensic efforts; or other persons, and especially old people, while using the water-closet.

Hypertrophy of the left ventricle of the heart is present in considerably more than half the cases of cerebral hemorrhage. Richerand is said to have been the first writer who pointed out pathologically the intimate connection between encephalic and cardiac disease. He says: "The dissection of patients who have died of apoplexy (cerebral hemorrhage) has proved to me that the excess of force in the left ventricle of the heart is a more powerful predisposing cause of the disease than a large head and short neck—a state of body which is supposed by most physicians to indicate the apoplectiform conformation." (*Nosographie Chirurgicale*, vol. iii.) In a memoir read by Richerand before the Ecole de Médicine, he refers to the case of the illustrious Cabanis, who died of apoplexy (cerebral hemorrhage) caused by, or associated with, disease of the heart. The left ventricle was enormously enlarged or hypertrophied. Eight ounces of blood were effused into the ventricles of the brain, and this effusion had been so violent that the septum lucidum was torn through, and the surface of the thalami and corpora striata made rough and jagged. Malpighi and Ramazzini also died of apoplexy connected with hypertrophy of the heart. At a later period, Lallemand, Broussais, Andral, Bouillaud, Bertin, and Rochoux directed attention to the same subject. MM. Bertin and Bouillaud remark that "the majority of the patients in whom hypertrophy of the left ventricle of the heart is present will be found to exhibit symptoms of cerebral congestion, and that many of them will fall victims of disease of the brain." (Vide *Forbes Winslow on Obscure Diseases of the Brain*, etc., p. 470.)

An intimate connection also exists between cerebral hemorrhage and disease of the kidneys. In almost all the cases of this accident, which the author has related, these organs were in a morbid state. T. Jones found the kidneys extensively diseased in twenty-nine out of thirty-six fatal cases of cerebral hemorrhage, showing the high percentage of 80.5. In twenty-four of these cases, the kidneys were small, hard, and granular, with wasted cortical substance. In four instances they were large, soft, congested, and cystic, with old cicatrices on the surface. In the remaining case, one kidney was reduced to a mere trace, while the other appeared healthy. (Vide *New Sydenham Soc. Year-Book*, 1864, p. 93.)

A remarkable case of meningeal hemorrhage is reported in the New-York *Medical Record*, vol. i. p. 106. In it the extravasation was due to injury of the blood-vessels of the pia mater, inflicted by certain osseous or calcareous growths developed from the dura mater. Some of them were needle-shaped, and at the autopsy it was found that the visceral arachnoid had been pierced by them. The subject was a male, aged 26, of intemperate habits, and a patient in Bellevue Hospital. Such osteophytes must, then, be looked upon as the efficient cause of meningeal hemorrhage in some rare instances.

There are certain morbid states of the blood itself which promote the occurrence of cerebral hemorrhage. According to Andral and Gavarret, there is an essential connection between cerebral hemorrhage and a diminution of the fibrin of the blood with an increase of the red corpuscles. Rupture of an artery not unfrequently occurs as a sequel of renal or cardiac disease, when it is probably due in part to an altered condition of the blood itself, resulting from these diseases. (*Tanner.*) There are also some rare cases in which cerebral hemorrhage occurs during convalescence from typhus and other acute infectious disorders; likewise, during scorbutus. (*Niemeyer.*) In such cases, the lesion of the blood, with which each of these diseases is known to be attended, is probably an important factor in the causation of the cerebral hemorrhage. Furthermore, the author has, in a few instances, met with meningeal hemorrhage in pale, anæmic, and badly-nourished children, who, at the same time, had purpuric spots on the skin. Some cases of this sort will be related in the chapter on Infantile Apoplexy, so-called. (Chapter VIII.) See cases LI., LII., LIII., and others belonging to that chapter.

In the following instance, profuse meningeal hemorrhage was probably induced by the injury resulting from a fall. The case was reported to the Pathological Society of Philadelphia, by Dr. John Ashurst, Jr. (Vide *American Journal of the Medical Sciences* for April, 1864, p. 427.)

A man, aged about 40, was brought to the Episcopal Hospital, about 3 P.M., October 16th, having fallen while getting off from the railway cars of the New-York line, in the morning. He had a deep cut through the buccinator and masseter muscles on the right side, penetrating to the bone. He was then (about six hours after the injury) in a state approaching coma, unable to articulate or swallow, but restless and uneasy. Dr. A. saw him about 10 A.M. the next day, (17th,) and found his condition as follows: Skin rather cool and clammy, and in the face very dusky, in some places almost blue; pupil of left eye dilated rather more than that of right eye, and both insensible to light; mouth slightly distorted; all the left side of the face seemed flabby and relaxed; the jaw was dropped; the tongue was stiff and very dry; it could not be protruded; breathing stertorous; slight palpebral but no orbital ecchymosis; urine had been passed spontaneously; there was some bleeding from the right ear when admitted, but it had ceased. From the symptoms and history of the case, Dr. A. suspected fracture at the base of the skull; but the autopsy showed this to be incorrect. No marked change occurred till the patient died about 8 A.M., on the 18th, nearly forty-eight hours after the injury.

Autopsy, seven hours after death.—Rigor mortis strongly marked; no external evidence of injury except the wound mentioned above.

On opening the skull, a considerable quantity of fluid blood escaped. Between the membranes and the brain, pressing on the base of the latter, on the right side and posteriorly, was a clot the size of a pigeon's egg or larger. There was also much uncoagulated blood bathing the base of the brain, and the membranes were exceedingly congested. Altogether not less than four to six ounces of fluid blood had been poured out upon the brain.

The thoracic viscera appeared healthy; some old adhesions existed in the right pleural cavity. A long fibrinous clot occupied the right ventricle of the heart, and extended into the pulmonary artery.

The liver was enlarged, and gave evidence of fatty degeneration; this, with the fact that a whisky-bottle was found in the patient's pocket, made it probable that he was a drinking man. The gall-bladder was distended. The spleen was lobulated but healthy. The right kidney enlarged and congested; the left of the usual size and healthy. No other abnormal appearances were observed.

Comments.—It is highly probable that the meningeal hemorrhage which occurred in this man's case had its origin in the commotion or contusion of the pia mater and of the surface of the brain which resulted from his fall. It is, however, barely possible that this hemorrhage began spontaneously while he was standing on the platform of the car, and that it was the hemorrhage itself which caused him to fall to earth. This case is here presented for the purpose of showing that an injury in the nature of a fall which, under ordinary circumstances, would prove to be but trivial and unimportant, may serve as the exciting cause of cerebral hemorrhage in a person who is predisposed to the occurrence of that disorder, from habits of alcoholic intemperance, or from degeneration of the cerebral blood-vessels.

Cerebral hemorrhages occur at all seasons of the year; occasionally, without any known cause, cases accumulate remarkably. They have also been observed at all hours of the day, and statistical tables have been made showing the comparative frequency of their occurrence at morning, midday, and evening. Although advanced age furnishes the largest number of cases, they occur at every period of life, and are met with even among children. Men are somewhat oftener attacked than women. (*Niemeyer.*)

But extravasation of blood into the brain is especially apt to occur when several of the causes mentioned above, such, for example, as abnormal fragility of the cerebral arteries, a plethoric state of the vascular system, and hypertrophy of the heart, happen to coöperate.

The occurrence of convulsive movements, especially when they are general and severe, may exert a powerful influence in the production of cerebral hemorrhage, as several of the cases which we have related abundantly show. Epilepsy and eclampsia will obviously be much more likely to induce cerebral hemorrhage when they occur in persons whose cerebral arteries are weakened by disease.

Anatomical Appearances produced by Cerebral Hemorrhages in general.—The extravasations are denominated capillary hemorrhages, and hemorrhagic clots, according as the bleeding consists of numerous small, closely-packed effusions, or of coagulated collections of extravasated blood having at least some considerable size.

In capillary hemorrhages, the cerebral substance appears dotted with dark-red punctate effusions at some spot of variable size. The cerebral substance between these minute extravasations either retains its normal color and consistence, or acquires a yellow or reddish hue to a variable extent from imbibition; it is relaxed and moist, or, lastly, it is broken down to a red pulp by the extravasation, and is then said to have undergone red softening. (*Niemeyer.*)

Small hemorrhagic clots sometimes press the brain-filaments apart; but larger ones break down the brain-substance and become mixed with it. In the former case, the effusion is sometimes elongated in the direction of the filaments; in the latter, it is more roundish or even irregular in shape. In the former case, the walls of the effusion are to some extent smooth; in the latter, they often appear ragged, and are frequently surrounded, for a space some lines thick, by a broken-down pulpy brain-substance discolored with blood. The size of the clot varies from that of a hemp-seed to that of the fist. If the extravasation occurs in the vicinity of a ventricle, it often breaks through the wall of the latter, and flows therein. Extravasations forming near the surface of the brain not unfrequently break through the cortical substance and escape into the subarachnoid space. Usually there is only one hemorrhagic effusion in the whole brain, occasionally several. The most frequent seat of these effusions is the corpus striatum, the thalamus opticus, and the large medullary masses of the cerebral hemispheres; less frequently they occur in the cortical substance of the cerebrum, in the cerebellum, and in the pons Varolii. Extravasations in the corpora quadrigemina, and in the medulla oblongata, are rare; and they hardly ever occur in the corpus callosum and fornix.

The contents of a recent hemorrhagic cavity consist of blood and broken-down brain-substance. The blood either remains fluid or becomes partly coagulated, and then the fibrin is occasionally deposited at the periphery, while the middle of the clot consists of fluid blood. Changes in the constituents and in the walls of the clot soon begin. The fibrin of the blood and the portions of brain mingled with the effusion break down into a detritus, the effusion itself becomes more fluid, the dark-red color becomes brown, then saffron-yellow. Granular pigment, and often also hæmatoidin crystals, are formed from the hæmatin. At the same time, in the immediate vicinity of the clot, there is a new formation of connective tissue starting from the neuroglia, which develops into a thick, hard layer or capsule, that envelops the clot. In the same way there occurs a new formation of delicate connective tissue, colored yellow by the pigment contents and serous infiltration, which covers the walls and traverses the clot as a fine network. After the elements of the extravasation have broken down, they slowly disappear, while their place is gradually supplied by serum; and when the transformation has become complete we find in the brain a cavity filled with clear liquid, surrounded by a callous substance, and covered and traversed by delicate yellow-colored connective tissue, a *so-called apoplectic cyst.* These cysts usually remain permanently. But occasionally the serum becomes absorbed, the walls approximate, and finally are separated only by a stratum of pigment. These callous spots, enclosing pigment striæ, are called *apoplectic cicatrices.* The cicatrization of a hemorrhagic effusion in the cortical substance is somewhat different. The effusion of blood under the pia mater, which is usually flat and extended, undergoes the same changes as the ingredients of a central clot. The red pulp gradually becomes a reddish-brown or saffron-yellow crumbly mass, which is bounded below by callous brain-substance, above by the pia mater. Lastly, we find an excavated pigmented plate, above which a serous effusion fills the cavity, resulting from the depression. These terminations of cerebral hemorrhage must be regarded as the most favorable ones. For, in some cases the reactive cerebritis in the vicinity of the injured portion of brain is not limited to a new formation of connective tissue. Extensive destruction of the cerebral substance from inflammatory softening then occurs; or the clot itself becomes transformed into an *abscess of the brain.* (*Niemeyer.*)

If the extravasation has been considerable, the parts of the brain untouched by the hemorrhage are exsanguinated, and the more so the larger the extravasation; small capillary hemorrhages have no influence on the amount of blood in the rest of the brain. In large effusions the hemisphere containing the clot is particularly anæmic. The subarachnoid spaces also are empty, the convolutions flattened, and the furrows have disappeared. Since the uneven appearance of the surface of the brain, when normal, is to a great extent due to the presence of cerebro-spinal fluid and of vessels containing blood in the depressions between the convolutions, the surface of the brain appears very smooth and even when there is a large extravasation. The brain rarely endures the injury occasioned by a considerable extravasation without impairment of its general nutrition. The gradual diminution of mental power observed in almost all cases of well-marked cerebral hemorrhage corresponds to a general atrophy of the brain; which, according to the investigations of Türk, is associated with degeneration, extending into the spinal medulla, of the nerve-filaments that communicate with the seat of the hemorrhage.

If the blood be effused in the subarachnoid space, in cases of meningeal hemorrhage, we usually find it as a more or less thick layer spread over the surface of the cerebrum and cerebellum. If the arachnoid be not torn, of course we cannot wash off the extravasation by turning a stream of water on it. Usually part of the extravasation reaches the ventricles, and there, also, we find more or less blood. In hemorrhages between the dura mater and arachnoid, that is, in the cavity of the arachnoid, the extravasation not unfrequently collects on the tentorium and at the base of the skull, and thence extends into the vertebral canal. But on the convexity of the hemispheres we also find coagula which may be washed off by squeezing out the sponge over them. In both forms of meningeal hemorrhage, there is either simple flattening of the convolutions and anæmia of the brain-substance, or else the laminæ of brain-substance next to the extravasation are suffused with blood and softened. (*Niemeyer.*)

With regard to the anatomical appearance due to the age of the clot: When effusion has taken place into the substance of the brain, if the patient has died during the attack, or shortly after, the hemorrhagic cavity is found filled with fresh-looking, dark-red, half-coagulated blood, its walls irregularly softened, and dyed to the extent of some lines deep with the coloring-matter of the blood; and a small stream of water directed upon this part at once removes the extravasated blood, and also a layer of softened cerebral matter. Again, if the patient has survived a week, the blood is found coagulated and the serum set free; but the presence of the clot has caused inflammation, so that the walls of the cavity are not only discolored, but more decidedly softened, and are softer in proportion as they are nearer the clot. If life be prolonged till the fifteenth day, the serosity is absorbed, but the walls of the cavity are still of a deep-red hue. About the seventeenth day, Virchow has discovered blood-crystals, or crystals of hæmatoidin, in the cavity. These blood-crystals were first discovered by Sir Everard Home, and have been more recently described and had their nature explained by Funke, Kunde, Lehmann, Beale, Parkes, and Sieveking. It appears that these crystals do not form from clotted blood until the blood-corpuscles have become ruptured by endosmosis. Their contents then escape and crystallize as the solution gradually becomes concentrated. (*Beale.*) Thus another guide to the age of the clot exists in the presence of these crystals. About the thirtieth day, if the patient lives so long, the clot is isolated and a membrane forms, at first muciform, fragile, intermixed with particles of cerebral matter, and also with some of the coloring matter of the clot. By degrees this membrane or capsule becomes more consistent. The clot also diminishes and some serum is probably secreted by the new membrane surrounding it. The cyst has been found fully formed, organized, and nearly empty by the thirtieth day. (*Macintyre.*) The cerebral walls surrounding the cyst, previously softened, now become indurated, and are stained yellow from the usual changes which the extravasated blood with which they are penetrated undergoes, a color, however, which they ultimately lose. The cavity thus formed may be filled at length with serum only; or, the serum being absorbed, the membranous cyst may ossify, and may be converted into a bony tumor. At other times the opposite sides of the cavity unite by a delicate connective tissue, which thus forms a species of cicatrix, but possessing so little power of conducting nervous influences that the patient seldom recovers from his palsy. Such is a short outline of the sequels of hemorrhage into the substance of the brain. (*Aitken.*) The size of a hemorrhagic cavity varies from a barley-corn to that of an egg, and their number is about as variable as their extent. Generally we find but one, sometimes two, and in a very few instances three or more cavities. When many hemorrhagic cavities exist in the brain it is rare to find them all of the same date. Some are old and almost obliterated, others are fresher, and others again quite recent, their different stages marking a distinct and different period of attack. Dr. Fuller has related a most interesting case of this nature, in which there were six clots, each of a different date, and in a different stage of discoloration, and corresponding to six well marked attacks of cerebral hemorrhage that had occurred in the course of nine months. (Vide *Diseases of the Chest*, p. 602.) This case is mentioned by Dr. Aitken.

According to statistics, the extravasation occurs in the cerebrum, and, for the most part, in the corpora striata and optic thalami, or their vicinity, in seven eighths of all the cases of cerebral hemorrhage, (*Niemeyer*,) as already stated.

Symptoms and Course of Cerebral Hemorrhage.—A brief account of the phenomena which were present in some of the cases related by Trousseau will give a better idea of the symptoms of this disorder than many pages of general description.

1. A very intelligent woman, aged 49, was enjoying excellent health when she noticed, one morning about 8 o'clock, an impediment in

her speech, and some numbness of her arm and leg. She walked down-stairs from the third floor and went to a neighboring chemist's shop. There she took a few drops of ether, and returned home, but with less facility, feeling the numbness rapidly increasing. On reaching the bottom of her stairs she was unable to go any further, and tried to save herself from falling by leaning against the wall, but dropped down, nevertheless, without losing consciousness or even feeling giddy. Her neighbors brought her to the Hôtel-Dieu. She had paralysis of the right side, (hemiplegia.)

2. Another woman had just prepared and served the family dinner at 4 o'clock P.M. She was eating with good appetite, and without headache or any other premonitory symptom. Suddenly she finds that she can not cut her bread; she says so to her husband, but with a thick voice. She tries to get up, and falls down with her chair, but without losing consciousness or feeling giddy. As she is raised up she is found to be hemiplegic, and on admission to hospital she herself relates the above details with perfect clearness.

3. A man, aged 63, while at breakfast, suddenly found some difficulty in holding his fork, and felt slightly giddy. On attempting to speak his tongue was observed to be thick. He staggered as he rose, felt weaker on one side than the other, but, with the help of his son, managed to walk to his bedroom. His intellect was perfectly clear. The hemiplegia made rapid progress, however, and became complete within half an hour. The intellect got clouded gradually more and more, and in three quarters of an hour he was in apoplectic stupor. Things went on from bad to worse, and he died comatose in the night.

4. A man, aged about 62, noticed, while at dinner, that one of his hands felt heavy. He faltered in his speech, but was not giddy. He tried to rise from his chair, but one of his legs having become paralyzed, he fell down, without losing consciousness, however. His children lifted him up, and with their assistance he walked to the next room, and there sat on a chair. Trousseau saw him three quarters of an hour after the manifestation of the first symptoms. He still retained his intellect. He answered questions to the point, although his speech was very much affected; and his left arm and leg were almost completely paralyzed as to motion. Profound coma set in a few hours later, and death occurred the following morning.

5. A woman, aged 56, and in as good health as ever, noticed while on her way home from marketing that she dragged her right leg, and that her right arm felt heavy. She even changed to her left hand a folded newspaper which she was carrying home, for fear of dropping it into the mud. She walked up-stairs to her room, undressed herself, and went to bed To questions of her husband she replied in a faltering voice. The

symptoms grew hourly worse. She became completely hemiplegic and partially unconscious toward evening. From about twelve hours after the first symptoms, and for three days, she lay in a state of profound stupor. At the end of that time she came out of it, but remained paralyzed on her left side for life.

6. A woman, aged 63, suddenly faltered in her speech, and was seized with weakness in one half of her body. There was no loss of consciousness, and no giddiness. She became hemiplegic, but ultimately recovered in great measure. The next year, however, she had another attack of cerebral hemorrhage. This time she was found in her bed in a state of profound coma. She died without having been roused; and at the autopsy there was found, in addition to the remains of the small hemorrhage of the previous year, an enormous clot, beginning in one optic thalamus and distending both lateral ventricles. (Vide *Lectures on Clinical Medicine*, vol. i. pp. 4–8.)

These cases serve to illustrate the symptomatology of the varieties of cerebral hemorrhage which are most frequently met with, and of those also which possess most importance in a practical point of view. It will be observed that the symptoms of paralysis were gradually developed in every instance where the attack occurred in the presence of witnesses.

Premonitory Symptoms of Cerebral Hemorrhage.—This topic possesses great practical importance. Cerebral hemorrhage is one of the gravest of diseases, and therefore it is all the more desirable to anticipate or prevent its occurrence. Now, the physician will doubtless succeed much better in doing this if he is well acquainted with its premonitory symptoms than if he is not. Moreover, after the extravasation has occurred, art can do but little, comparatively speaking, for the relief of the patient. To the author, this topic seems not to have received sufficient attention at the hands of classical writers. He therefore proposes to discuss it more fully than is usual, and to give as thorough an account of it as he can, with the aid of what others have recorded.

There seems to be considerable difference of opinion as to the frequency with which premonitory symptoms occur in cerebral hemorrhage. Some think that in most cases this disease is not attended with any warning signs. Forbes Winslow, on the other hand, holds that it is almost invariably preceded by some warning sign, and the author coincides in this opinion. It is, however, probable that sometimes the premonitory symptoms are so slight as to entirely escape the patient's notice, and that, in other cases, their occurrence has been forgotten by the patient on account of the shock which his memory has received from the extravasation itself. But in many instances the premonitory symptoms are such as to excite in the physician, and even in the laity, apprehension that an attack of palsy or apoplexy is threatened. Such patients often complain of severe vertigo and headache, or of heaviness and fulness in the head, of confusion of the intellect, of noise in the ears, and of flashes before the eyes. They sleep badly, and are excited or irritable. Temporary feelings of formication and numbness in certain limbs, momentary loss of memory for some words and figures, and temporary paralysis confined to certain groups of muscles, are peculiarly ominous signs. Let us, however, devote a little time to considering these warning symptoms of cerebral hemorrhage in detail, and in the light of recorded experience, for it can not fail of proving both useful and interesting.

1. *Of the Warnings which are Sensorial.*—Forbes Winslow says: "Apoplexy [cerebral hemorrhage] is almost invariably preceded by either severe vertigo, noises of some kind in the head, confusion of intellect, or severe paroxysms of cephalalgia." (Vide *On the Obscure Diseases of the Brain*, etc., p. 402.) Again, vertigo is exceedingly frequent at an advanced period of life, and often indicates the approach and formation of disease in the brain. But it may be only sympathetic or due to reflex irritation. Cerebral vertigo, however, is easily recognized by the absence of those affections of other organs which sympathetically disorder the brain, such as gastric, renal, and hepatic derangement, loss of blood, and long-continued exhausting discharges.

Andral relates a case of apoplexy in which a complete loss of sensation was observed some time previously to the attack in isolated parts of the thorax. There existed five or six portions of skin, about the size of a five-shilling piece each, which showed no signs of sensibility, even when pinched or pricked with a sharp instrument. In other parts of the thorax the sensibility of the skin continued intact. These circumscribed states of cutaneous insensibility were not continuous in their manifestation, sensibility and insensibility appearing to be alternate conditions. (*Ibidem*, p. 404.)

"In some apoplectic cases there has been observed a decided impairment of sensation in one half of the mucous membrane of the nasal fossæ." (*Ibidem*, p. 404.) "In other cases the anæsthesia has been confined to a side of the face, one of the fingers, to the scalp, and in some remarkable cases the feeling of numbness has been restricted altogether to a lateral half of one of the fingers." (*Ibidem*, p. 405.)

"Slight degrees of cutaneous anæsthesia have existed for many years previously to fatal attacks of apoplexy and paralysis. In some instances this diminution of sensibility has been associated with a feeling of intense cold in one of the extremities. Andral, Romberg, and other pathologists have noticed this incipient symptom of apoplexy, paralysis, and softening." Again, among the premonitory signs of cerebral hemorrhage Andral observes odd sensations, resembling a feeling of intense cold, confined to the tips of the fingers. The ends of the fingers," he says, "appear as if they had been plunged into iced water." Dr. Cooke refers to a case of apoplexy where the patient complained, some weeks before the attack, of a painful sensation of cold in one of the feet. There was no apparent diminution of sensibility in the leg nor any other part of the body. (*Ibidem*, pp. 404, 405.)

Derangement, perversion, and aberration of sight, says Forbes Winslow, will be found more frequently symptomatic of disease of the brain than impairment, loss, or exaltation of this function. Portal predicted an attack of apoplexy in a gentleman (apparently at the time in perfect health) from observing a slight fixedness of his left eye, and a trifling weakness (incipient paralysis) on the same side of the body. Apoplectic patients have been known to complain, before an attack, of objects appearing to be red. Others fancy that a line of a similar tint borders all things they see. They also complain of a sensation like that experienced by the eyes when they have been exposed for some time to a strong light. Objects appear as if they were dotted with black or red spots, or the patient imagines that a mist or thin veil intervenes between them and the eyes. When speaking of the disorders of vision which precede or accompany the occurrence of cerebral hemorrhage, Andral remarks: "Sight is sometimes, but not always, disturbed. We see individuals struck down with apoplexy and affected with paralysis and loss of sensation, where, nevertheless, consciousness and vision remain. Different sensations, resulting from disturbance of this function, are experienced by patients, who describe them in different ways; some say that they have motes before their eyes; others, that they see the light as through a cloud, just as on the onset of cataract—yet here the crystalline lens is clear; others see various colors. Sometimes those who, at a later period, are attacked with apoplexy, [cerebral hemorrhage,] have the sight modified for a longer or shorter time before the attack in such a manner that *all objects appear double*—a symptom which is sometimes transient, being present one day and not on another. In other cases the loss of sight is nearly complete, but such cases are very rare. When the sight is lost, this may take place on one side or on both; and this blindness coincides with the loss of numerous other senses." (Vide *Forbes Winslow on the Obscure Diseases of the Brain*, etc., pp. 434–439.)

Sudden loss of sight is occasionally a premonitory symptom of apoplexy. Andral relates the following case: "A locksmith experienced considerable vertigo for eight days. He then became blind. He remained in this state of vision for fifteen days, when he was seized with sudden loss of consciousness, followed by paralysis. His sight was gradually restored, but the hemiplegia continued." But total loss of sight, unaccompanied by any other signs of brain-disease, may exist for a still longer period before the symptoms of apoplexy occur. For example, Baron Hornestein, whose case is cited by Wepfer, became blind three weeks before he was seized with a fatal attack of apoplexy. (Vide *Forbes Winslow on the Obscure Diseases of the Brain*, etc., p. 426.)

Hyperæsthesia, or exaltation of vision, is occasionally observed among the premonitory symptoms of cerebral hemorrhage. Forbes Winslow refers to a case in which this symptom was precursory of the attack for at least ten days. It was, however, associated with severe vertigo. Dr. Breschet relates that, when he was interne at the Bicêtre in 1811, the infirmier of the surgical ward one day astonished him by the capacity which his vision had acquired since the previous day. He could distinguish the most minute objects at an enormous distance. Five hours afterward he felt a slight headache, and in a few hours more was seized with cerebral hemorrhage, ("*une apoplexie foudroyante*") and died the next night. A recent coagulum of blood was found in the right optic thalamus. The inflammation which preceded this effusion had affected a part of the brain immediately concerned in vision. (*Ibidem*, pp. 429–431.)

Strabismus suddenly appearing, and associated with acute headache, mental confusion, or severe vertigo, has occasionally been found precursory of cerebral hemorrhage, and often denotes the commencement of effusion. A gentleman, who had complained for a few days of headache and depression of spirits, was observed, whilst at dinner, to have strabismus. A few minutes subsequently he had a fit of apoplexy. (*Ibidem*, p. 445.) In this case the squint appears to have been the first symptom which was produced by the extravasation of blood in the brain.

Antecedent to attacks of cerebral hemorrhage a curious *phantasm* is occasionally observed. A spectral image resembling the patient is noticed. This form of hallucination is termed *deuteroscopia*. The phenomenon is considered of rare occurrence, even among the insane. Aristotle refers to this type of illusion. He says that a certain Antipheron, when he was walking, saw a phantasmal reflection of himself advancing toward him. (*Ibidem*, p. 441.)

Attacks of apoplexy, paralysis, cerebral hemorrhage, etc., have often been preceded by *double vision*. Dr. Gregory was acquainted with a sportsman who, one day when out shooting, disputed with his gamekeeper as to the number of dogs they had in the field. He asked how he came to bring so many as *eight* dogs with him. The servant assured him that there were but *four*, and the gentleman, at once aware of his situation, mounted his horse, and rode home. He had not been long in the house when he was attacked by apoplexy and died. (Vide *Sir Thomas Watson's Practice of Physic*, vol. i. p. 414, Am. ed. 1872.)

Disordered Hearing.—Occasionally, among the incipient signs of cerebral disease, we find sudden paralysis of the auditory nerve. These cases, however, are not of common occurrence. The symptoms most frequently noticed in insidious affections of the brain, so far as the sense of hearing is concerned, are a gradual impairment or a sluggish state of that faculty, on the one hand, and a hyperæsthesia or morbid exaltation of it, on the other. (Vide *Forbes Winslow on the Obscure Diseases of the Brain*, etc., p. 446.)

The Taste, *Touch*, *and Smell* also occasionally exhibit, at the commencement of cerebral disease, evidence of impairment, exaltation, and perversion. The tactile sensibility is not unfrequently disordered in the forming stage of paralysis. The patient complains of a feeling of numbness and want of sensibility in the ramifications of the tactile nerves at the ends of the fingers. This disorder of sensation often precedes attacks of general paralysis. Sir James Y. Simpson has heard patients having incipient general paralysis complain of their fingers "feeling like sausages." A tailor, who died of this disease, lost all sensation at the tips of his fingers for twelve months before any suspicion arose as to the healthy state of his brain. He was unable to work with his needle, as he never knew, owing to the anæsthesia, when he had it between his fingers. (*Ibidem*, pp. 448, 449.)

2. *Of the Warnings which are Motorial.*—"In cases of incipient paralysis from cerebral hemorrhage," says Andral, "the patients perceive that one of the extremities has less strength than the other; one of the hands can hold objects less strongly than the other; one of the arms appears insensible to them, or the patient's legs drag a little in walking." He continues—and the remark is of great practical importance: "This commencement of paralysis *may remain stationary for a long time*, then it is seen to progressively increase, or else it becomes all at once more considerable." Patients so affected are at the beginning frequently observed to drop things from the hand. This is the first symptom of impaired motility that may be observed for some weeks prior to an apparently sudden and acute attack of cerebral hemorrhage followed by hemiplegia. Furthermore, the loss of motor power in cases belonging to this category is occasionally confined to one of the fingers, this being the only appreciable symptom to excite alarm; and thus a limited paralysis of this kind sometimes occurs as one of first symptoms threatening cerebral hemorrhage. Again, inability to hold the pen when writing, to handle the razor with steadiness when shaving, (from impairment of muscular strength in the fingers,) and to play the piano with the usual vigor and facility, have been observed among the first warnings of hemiplegia and cerebral hemorrhage. (Vide *Forbes Winslow on the Obscure Diseases of the Brain*, etc. pp. 333, 334.)

Occasionally, the tongue is observed to be tremulous with its tip turned slightly to one side. This symptom has sometimes been observed as the *avant courrier* of cerebral hemorrhage, softening, and paralysis. (*Ibidem*, p. 345.)

Among the early symptoms of cerebral hemorrhage is a slight degree of facial paralysis. Occasionally it affects the eyebrow as well as the mouth. The patient has, to some extent, lost power over one of his eyelids, and it seems to be too heavy to be raised. One eyebrow is also more elevated than the other. Not unfrequently the mouth is seen to be drawn somewhat to one side, that is, upward and outward toward the corresponding ear.

An inability to forcibly eject saliva from the mouth, or to spit as usual, in consequence of the orbicularis oris and buccinator muscles being slightly paralyzed, has been one of the first symptoms produced by cerebral compression. (Vide *Forbes Winslow on the Obscure Diseases of the Brain*, etc., p. 345.)

The patient is sometimes seen to oscillate in walking like a drunken man. He has partially lost his balancing power. In cases of impending paralysis [and cerebral hemorrhage] this symptom is often observed. The gestures, gait, and walk closely resemble the movements of a person slightly under the influence of alcoholic stimulants. (*Ibidem*, p. 337.)

In some cases the patient will be heard to complain, for some time before an outbreak of cerebral hemorrhage, of suffering from a *spasmodic affection* of the muscles of the leg and arm, but particularly of the former. In other instances the legs are stiff, and show a want of suppleness, independently of any loss of sensibility, or any impairment of muscular power. These symptoms often precede paralytic attacks, but they are generally associated with other characteristic evidences of cerebral mischief. Occasionally, the spasm seizes the whole of the leg, which becomes quite *tetanic*. This symptom, observed in the early stages of acute cerebral irritation, is connected in some cases, but not always, with organic disease, such as inflammatory softening of the brain. A sensation of slight stiffness of the limbs, combined with pain, analogous to that of rheumatism, spasm, and convulsive twitching of the muscles, if accompanied by headache, mental confusion, vertigo, etc., should always receive careful attention from the medical practitioner. (*Ibidem*, p. 335.)

Dr. Cooke says: "I have known a person first lose the strength of his legs, then talk childishly, fiddle with his knife and fork during dinner, to the confusion of his family, attempt in vain to direct the morsel to his mouth, and at length carried to bed several hours before he became apoplectic." Besides, inability to swallow, from paralysis of the muscles concerned in deglutition, may sometimes be observed among the early symptoms of cerebral hemorrhage. (Vide *Forbes Winslow on the Obscure Diseases of the Brain*, etc., p. 334.)

Handwriting.—Some singular deviations from the usual character of the handwriting have been observed as the first signs of approaching general paralysis, cerebral softening, and cerebral hemorrhage. The patient has not been able to write in a straight line nor to form his letters correctly. Occasionally he misplaces his words in a strange manner, and appears to have lost all power of correct spelling; or in writing he wrongly substitutes one word for another, his letters are flighty, full of eccentricities, blunders, and erasures. How often have these symptoms been observed for months before a suspicion has been excited as to the soundness of the brain! (*Ibidem*, p. 346.)

Occasionally, in incipient disease of the brain, the patient is observed to make repeated but ineffectual efforts to utter articulate sounds. He is seen to open and close his lips as if trying to speak, but can not do so. The attempt thus made produces a singular movement of the lips, similar to that seen in the action of smoking a pipe, conveying to those who have observed the phenomenon the idea that the patient has a symptom described as symptomatic of dangerous cerebral disorder, and designated by French pathologists, "*Le malade fume la pipe.*" These symptoms of failing vocal power may exist for several months before the attention is directed to them. Such morbid affections of articulation are found among the most insidious signs of cerebral disease. (*Ibidem*, p. 376.)

Loss of voice, (*aphonia*,) accompanied by difficulty in swallowing, (dysphagia,) occasionally results from pressure upon or change of structure at the origin, or in the course of the hypoglossal and glosso-pharyngeal nerves. Dr. Copland relates a case of the kind in which these symptoms preceded a fatal attack of apoplexy some months. The patient, a gentleman, aged 50, had lost all power to utter articulate sounds for many months. He also swallowed substances with great difficulty, and sometimes was unable to do it at all, unless they were placed on the base of his tongue. He could not protrude nor move his tongue in the least. No other part of his body was paralyzed. He had neither headache nor any other ailment, and regularly attended to the duties of his profession during the usual hours of business, but was obliged to communicate what he wished to say by writing. (*Ibidem*, pp. 377, 378, note.)

Sometimes a *sudden loss of speech* is the first sign that an attack of cerebral hemorrhage and hemiplegia is impending. A gentleman, aged 35, mentioned by Dr. Abercrombie, suddenly lost his speech, while standing in the street conversing with a friend; he recovered it after a few minutes, walked home, and made no particular complaint of indisposition. That evening, however, he suddenly fell from his chair, speechless and paralytic on the right side, but without coma, being sensible of what was said to him, and answering by signs. He was then confined to bed for several weeks without any change of symptoms. At the end of three months he had recovered the motion of his leg so far as to walk a little, dragging forward the leg by a motion of the whole right side of his body. Afterward he improved considerably in strength, so that he could walk several miles; but his thigh and leg continued to be dragged forward by the same kind of effort, without any further improvement. He never recovered any degree of motion in the arm or hand; he could not move even the fingers; his speech was very inarticulate, and his countenance expressive of great imbecility. He continued in this state without relapse or any further improvement for fifteen years, and died, aged 50, rather suddenly, with symptoms resembling those of typhus, but without coma. (*Ibidem*, pp. 378, 379.)

Loss of speech, (*aphasia*,) followed in a few weeks or months by a fatal attack of apoplexy, has been known to occur without any previous symptoms of brain or nervous disorder, that is, without headache, vertigo, noise in the ears, loss of sensibility or motility, depression of the spirits, affection of the vision, or any other cerebral symptom of a suspicious character. Dr. Graves cites the following illustrative case: "A barrister was walking up and down the hall of the Four Courts waiting for a case to come on, and chatting with one friend and another. As the hall was rather crowded and hot, he went out into the area of the courts for the sake of the air, and had not remained there more than ten minutes when an old friend from the country came up and spoke to him. He was pleased to see his friend, and wished to inquire about his family, when he found, to his great surprise, that he could not utter a single audible sound; he had completely lost his voice. He recovered the use of his tongue in about three weeks, but not completely, for some slowness of speech remained. When the loss of speech was first perceived, his friend brought him home in a carriage, and during the day he had several attacks of vertigo, and afterward hemiplegia. For several hours, however, before distortion of the face or any of the usual symptoms of paralysis had commenced, the only existing symptom was loss of speech. This gentleman died of apoplexy in about two months." (Vide *Graves's Clinical Medicine*, p. 688, Dublin, 1843; also *Forbes Winslow on the Obscure Diseases of the Brain*, etc., p. 380.)

3. *Of the warnings which are psychical.*—Prior to attacks of apoplexy, paralysis, and softening of the brain, the patient not unfrequently complains of a stunned, inactive, confused, and sluggish state of the memory, indicated by a difficulty in recalling with facility ideas to the mind. The attempt, under such conditions of impending disease, to revivify former impressions and thoughts requires a strong effort, and is attended

with sensations of physical distress that are clearly referrible to the head. This mental impairment is often connected with cerebral hyperæmia. (Vide *Forbes Winslow on the Obscure Diseases of the Brain*, etc., p. 249.)

A disposition to excessive sleep, or a lethargic state, is not unfrequently a precursory symptom of apoplexy and cerebral hemorrhage. (*Ibidem*, p. 459.) *Great depression of spirits* is sometimes a premonitory symptom of these diseases. (*Ibidem*, p. 192.) *Mental excitement*, on the other hand, is often found to be a precursory sign. For some days prior to the attack, the patient has been observed to exhibit symptoms of unusual irritability and irascibility. (*Ibidem*, p. 188.)

Frightful dreams and the severer forms of *incubus or nightmare* are sometimes complained of by patients before they are attacked with apoplexy, as well as cerebritis and insanity. A patient, referred to by Forbes Winslow, had for a fortnight preceding an attack of apoplexy a consecutive series of horrible dreams, in one of which he fancied that he was being scalped by Indians. Others have dreamt of falling down precipices, and of being torn to pieces by wild beasts. (*Ibidem*, p. 461.)

A sudden revivification or improvement of the memory occurring to persons in advanced life is occasionally the precursor of death or an attack of fatal apoplexy. Hippocrates noticed this phenomenon. A disposition to talk garrulously about events that apparently have long been forgotten is also sometimes observed among the incipient symptoms of cerebral hemorrhage and paralysis. (*Ibidem*, p. 283.)

"*Sudden and transient attacks of forgetfulness*, if associated with an inability to articulate correctly, are most grave and important symptoms when considered in relation to a questionable state of the brain. These temporary and apparently trifling conditions of impaired memory and defective speech are often the preludes to serious cerebral disease,—the dark and threatening clouds that occasionally envelop, obscure, and often eclipse the mind previously to fatal attacks of paralysis, softening, apoplexy, and insanity." The same writer quotes the following case from Andral's *Clinique:* "A man, about 50 years of age, forgot his own name. He was from time to time convinced that he was dead. He no longer recognized his immediate relatives. He continued fifteen days in this state, when he died of an attack of apoplexy, [cerebral hemorrhage.] The *post-mortem* examination revealed an extravasation of blood within one of the hemispheres of the brain. There was no other important cerebral lesion." (Vide *Forbes Winslow on the Obscure Diseases of the Brain*, etc., pp. 250, 251.)

Dr. Rush cites a case of paralysis in which the premonitory symptom was *forgetfulness* how to spell the most common and familiar words.

A man aged 57, who up to that time had led a grave and even an austere life, unexpectedly abandoned himself to the pursuit of amusements that were childish and unsuited to his age. A few months afterward he was suddenly seized with cerebral hemorrhage (*apoplexie foudroyante*) and his life destroyed. (*Ibidem*, p. 202.)

Involuntary articulation, or thinking aloud.—A distinguished physician observed this symptom to precede an attack of paralysis in the case of a nobleman who for many years was Prime Minister of Great Britain. Moreover, the patient is observed to talk to himself, in many conditions of cerebral irritation as well as in structural disease, and the commencement of insanity is often detected by this symptom. I am fully aware, says Forbes Winslow, that this eccentric habit is quite consistent with a perfect state of health of body and mind; but, nevertheless, it is a symptom that should be carefully regarded in all cases of *suspected* disease of the brain coming on suddenly, at an advanced period of life, particularly if conjoined with other signs of cerebral disorder. (Vide *On the Obscure Diseases of the Brain*, etc., pp. 391, 392.)

"We can not," says Portal, "hear without astonishment the remarks sometimes made by those who are threatened with attacks of apoplexy. All their senses appear perfect and entire, but their minds appear to have acquired an inspired and prophetic power. Their first impression is that they are about to quit the world. Then they predict the future by the present, and the event justifying the prediction, they are regarded as true prophets." (*Ibidem*, p. 160.)

Having discussed the premonitory signs of cerebral hemorrhage at considerable, but not at unnecessary length, we will now proceed to describe, with some minuteness, the symptoms which are produced by the extravasation itself. When the brain-filaments are broken down by large extravasations, or softened by capillary hemorrhage, and the patient survives the accident, the result which is generally seen is that the muscles on the side of the body opposite to the seat of the extravasation are more or less extensively paralyzed, that is, a more or less complete state of hemiplegia is produced. Small hemorrhages, however, sometimes escape recognition during life because they do not produce any paralysis, and we can readily understand the reason if we bear in mind that certain parts of the brain, particularly the medullary substance of the cerebrum, may be destroyed without producing any disturbance of function that is perceptible.

We have elsewhere designated the corpus striatum and optic thalamus as the most frequent seat of hemorrhage; a destruction of these parts, or of the pedunculi cerebri, induces extensive paralysis in the opposite half of the body. We may readily determine that the paralysis, resulting from destruction of the above-mentioned parts, depends only on interruption of the conduction between the organs acting in thought and will, and the motor nerves and muscles; the power to think and will is unimpaired.

After the hemorrhagic seizure has become fully developed, if we ask the patient to give us his paralyzed hand, he shows his desire to fulfil the request by taking this hand in the other one, whose muscles and nerves are under the control of his will, in order to accomplish the act. Furthermore, in all cases of cerebral hemorrhage that are recent, every motor nerve on the paralyzed side, to which we apply the induced current of electricity, causes contraction in the muscles supplied by it. Hence, the only failure is in the communication between the central excitory apparatus and the motor nerves. This interruption has no effect on those movements of the paralyzed side which occur in a reflex manner without the influence of the will; for patients who, as a result of extravasation on the left side of the brain, cannot move the right arm or leg, move the right side of the thorax just as well as the left during respiration. And the connection between the motor nerves and those brain-filaments and ganglion-cells which are excited during certain states and feelings of the mind is not always destroyed with the interruption of conduction at present under consideration. This is shown by the fact that some patients who cannot make the motions of laughing or crying on the paralyzed side of the face, at our request, can do so when they do not *will* to do it, and when their feelings spontaneously lead them to express themselves in that way. Likewise, the interruption in conducting the impulses of the will, from the central organs to the motor nerves, does not necessarily imply that the communication between the latter and the sensory and the other motor (reflex) filaments is interrupted. On the contrary, we sometimes find that the reflex movements remain undisturbed in the paralyzed parts, or that they occur even more readily than before, so that it appears as if, when the excitement of the motor nerves is no longer subject to the will, they occur more readily than they otherwise would. (*Niemeyer.*)

Paralysis of half the body or hemiplegia, when due to destruction of the corpus striatum and thalamus of one hemisphere, is characterized by its limitation to the muscles of the extremities, to the muscles of the face which belong to the angle of the mouth and nose, and to the muscles that protrude the tongue. Such patients can almost always chew normally on the affected side, wrinkle the forehead, open and shut the eyelids, move the eyes in any direction, etc. But, at the same time, the mouth hangs down on the affected side, the nostril is contracted, and occasionally the paralyzed cheek flaps about like a loose sail at every expiration, while on the sound side the angle of the mouth is drawn upward and outward toward the ear, and the nostril is dilated. If the patient protrudes his tongue, its point deviates toward the paralyzed side, because the muscles of the sound side only are concerned in the act of pushing forward the root of the organ and elongating it. At the same time also the patient cannot lift the paralyzed arm and leg an inch from the bed. In most cases this motor hemiplegia is accompanied with anæsthesia of the same parts of the body; but, in a short time, this symptom usually passes more or less completely away. This course of the anæsthesia, as well as the experience that animals have no sensation immediately after destruction of their corpus striatum and thalamus, and that some time after destroying these parts the power of feeling peripheral pain returns, appears to indicate that the temporary anæsthesia of the paralyzed half of the body does not depend immediately on destruction of the corpus striatum and thalamus, but on compression of the capillaries in the parts of the brain lying below them caused by the effusion of blood. (*Niemeyer.*)

The same symptoms as are caused by extravasations into the thalamus and corpus striatum, are induced by extravasations in other parts of the cerebrum, provided they are extensive enough to compress the capillaries of the thalamus and corpus striatum. The only difference is the following: A large hemorrhagic clot, destroying the corpus striatum or thalamus, leaves a hemiplegia that never disappears; but a small clot in these parts, by which the nerve-filaments and ganglion-cells are not broken down, but only pressed apart, leaves paralysis which occasionally is temporary. Hence we may conclude that the apparatus for exciting the motor nerves, which doubtless exists in the brain, although it may itself be excited by the will, is located in the vicinity of the corpus striatum and thalamus. On the other hand, extensive hemorrhagic clots in other parts of the cerebrum (for example, in the medullary substance above the ventricles) not unfrequently leave hemiplegia which sooner or later disappears again. From this course we may suppose that the capillaries of the motor centres being relieved from pressure by the partial absorption of the extravasation, have again become permeable to the blood; or that the collateral œdema in the vicinity of the broken-down part of the brain, which at one time extended to the motor centres, has disappeared with the cicatrization of the hemorrhagic cavity. (*Niemeyer.*)

Hemiplegia is present in some, but not all of the cases where the effusion occurs in the cortical subtance of the cerebrum; such extravasations are generally accompanied by hemorrhage into the subarachnoid connective tissue. This difference with regard to the symptoms of paralysis doubtless depends on how far the oft-mentioned results of the hemorrhage, compression of the capillaries, or collateral œdema, extend inward; whether they reach the corpus striatum and thalamus or not. When the clot is in the above-mentioned position, general convulsions are often observed, and in most cases there is severe disturbance of the psychical functions. But, since experience shows that persons with far advanced and extensive degeneration or atrophy of the cortical substance, (if it be limited to one side,) often have no psychical disturbance, the frequent occurrence of the latter, in cases where the extravasation occurs on only one side, is probably due to the fact that the hemorrhage readily affects the other hemisphere, and particularly because this is likely to occur from the consecutive inflammation of the pia mater and arachnoid, which has a strong tendency to spread. (*Niemeyer.*)

Hemorrhage in the pons, if considerable in size, and in the medulla oblongata,[1] even if very slight, usually causes death. In cases where small effusions occur in the lateral portions of the pons, there are anæsthesia and motor palsy on the opposite side of the body; when they occur in the middle of the pons, there is paralysis on both sides.

Hemorrhage in the cerebellum is often attended with paralysis of the opposite side. But this cannot depend on the affection of the cerebellum itself, for there is often no paralysis observed when this part of the brain is extensively destroyed.[2] (*Niemeyer.*)

We must not think that because extravasations are found in very different parts of the brain, the different cases of paralysis depending on cerebral hemorrhage will differ widely from each other. On the contrary, the large majority of these cases show a great similarity, as they exhibit the symptoms of hemiplegia above described. It is, of course, of great practical importance to know this, which is simply explained by the fact that, according to statistics, seven eighths of all cerebral hemorrhages are located in the cerebrum itself, and chiefly in the corpus striatum or optic thalamus, or in the neighboring parts, as already stated. There are, however, some wonderful exceptions to the one-sidedness of the paralysis, and to its occurrence on the opposite side, in cases of hemorrhage in the cerebrum, which we are at present unable to satisfactorily explain. But, we must add, that of late, since all anomalies associated with the hemorrhage, particularly thrombosis and embolism, or plugging of the cerebral arteries, are more carefully attended to, and used in explaining the symptoms, the number of such cases published has greatly decreased. (*Niemeyer.*)

In occasional instances, cerebral hemorrhage produces the phenomena of apoplexy, properly so-called, as we have fully shown in the fore part of this chapter. Sometimes there is vomiting at the commencement of the attack, with a very slow pulse and contracted pupils. When the hemorrhage is profuse, and the intra-cranial space is considerably encroached upon by the effusion, a very important symptom is developed, namely, a remarkable pulsation or throbbing of the carotid arteries. This symptom is generally regarded as a sign of increased determination of blood to the head, although it really indicates that the flow of blood into the skull is obstructed; we may at any moment induce the same phenomena in the artery of the finger by tying a string tightly around the end of the finger. If we find this symptom when there is no hypertrophy of the left ventricle, nor corresponding pulsation in other arteries, it will, in doubtful cases, be a great aid to the diagnosis of some brain-disease encroaching on the cranial cavity. (*Niemeyer.*)

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[1] According to *M. Lévier*, nine cases of hemorrhage in the medulla oblongata have been reported, four only being pure; the results were, loss of consciousness, involuntary epileptiform movements, and sudden death. (Vide *American Journal of the Medical Sciences* for April, 1867, p. 531; also *Brit. Med. Journal*, Dec. 22d, 1866, and *Gaz. Méd. de Paris*, June 9th, 1866.)

[2] According to M. Hillairet, the course of hemorrhage in the cerebellum is gradual in some cases, but in others so rapid as to kill instantaneously. Vomiting is a special symptom, and so is loss of power in the limbs, not amounting to actual paralysis. Hemiplegia occurs in only a third of the cases; it is always crossed. Crossed facial paralysis, and deviation of the tongue are exceptional. Speech is generally drawling and slow. Sensibility is unaffected except towards the fatal close, and the same is the case with the special senses. Convulsions do not occur if the cerebellar lesion is uncomplicated. (Vide *New Sydenham Soc. Year-Book*, 1859, p. 203; and *Annuaire de Méd. et Chir. pratique*, par Jamain, 1859, pp. 39-63.)

The following case is presented for the purpose of illustrating the symptoms which are sometimes produced by hemorrhage in the pons.

CASE XXXI.

Extravasation of blood in the pons Varolii, terminating in recovery; intellect unaffected; reported by Dr. MÖLLER.

A female, æt. 24, was suddenly taken ill with pain in the left side of the head, and stiffness and numbness of the whole left half of the body, soon succeeded by paralysis. The facial and hypoglossal nerves were involved. There was difficulty of swallowing, but no loss of consciousness. The respirations were frequent and shallow; the pulse small, 140; the state of the heart normal. In a few hours, the paralysis disappeared, except that the sensibility remained impaired, especially in the face; the speech was also imperfect; the special senses were normal on both sides. After two days, another attack of collapse occurred, attended with a feeling of rigidity on the right side and paresis of the right arm. The morbid phenomena again gradually disappeared; the anæsthesia remained longest in the right hand, which continued also to be affected with slight tremor and choreic twitchings for a long time. The gait for a long time manifested derangement of the coördinating faculty, which, however, could be prevented when the patient directed specially her attention to the matter. Sobbing also was prone to occur. At last all that was left was a certain slowness of speech and movement.

Möller remarks that the pons is indicated to be the seat of the lesion, by the disorder so speedily affecting the two sides, one after the other, and by the maintenance of consciousness. From the existence and the long duration of impairment of sensibility it may be inferred that the posterior and upper layers of the pons were affected, which are specially traversed by the sensitive fibres. The derangement of the respiration and circulation he refers to an independent coincident pulmonary disorder, as he observes that such acceleration of the breathing and of the pulse have not occurred in any recorded case of disease of the pons. The derangement of coördination indicates that the cerebellum also was involved. (Vide *New Sydenham Soc. Year-Book* for 1864, pp. 88, 89; *Deutsche Klinik*, 49, 1863; *Schmidt's Jahrb.*, vol. 124, p. 24.)

The next case is presented for the purpose of showing the symptoms which are generally produced by moderate extravasation in a crus cerebri.

CASE XXXII.

Sudden paralysis of right side of body; intellect unaffected; death from broncho-pneumonia at the end of two months; autopsy; small coagulum in left crus cerebri, etc.; reported by H. WEBER, M.D.

A man, aged 52, affected with disease of the aortic valves, and rigidity of the arteries, was subject to headache and disturbed sleep during the last years of his life. About two months before death he was suddenly seized with paralysis of the right side of the body, producing almost total loss of motor, and considerable impairment of sensory power. At the same time the muscles supplied by the third nerve of the left side were also paralyzed. The uvula was drawn to the left; the right half of the palate was pendulous. He died of broncho-pneumonia, after slight but perceptible improvement in the phenomena of paralysis. The intellectual faculties were quite unaffected, as also the special senses. There was imperfect and less persistent paralysis of the muscles of the trunk, and of the fifth, of the portio dura of the seventh, and of the ninth cerebral nerves of the same side; the affection of the pneumogastric nerve manifested itself in the more than usually slow and irregular action of the heart during the first days after the seizure; and to the impaired action of this nerve, as well as of the sympathetic, Weber ascribes the disposition to the fatal pulmonary inflammation. The participation of the sympathetic was further evinced by the increased temperature of the paralyzed side.

At the post-mortem an oblong clot of blood was discovered in the internal half of the left crus cerebri, about 0.6 inch long and 0.25 inch broad, and almost as deep; it was situated very close to the internal and inferior surface, being separated from it only by a thin layer of nerve-substance; its commencement was immediately in front of the pons. Microscopically examined, the clot presented many shrivelled blood-corpuscles, besides many apparently unaltered ones. The surrounding yellowish and tense tissue (membrane ?) contained scarcely any nerve-fibres, but much connective tissue. In the left third nerve many oil-globules, and granules of various sizes, and also small granular corpuscles were found, which were absent in the right nerve; the fibres in the left nerve were scanty and broken down. All the other parts of the encephalon were normal.

No circus movements were observed in this case, nor in one cited from Andral, such as are produced in animals by section of one crus cerebri. In a case, however, put on record by Dr. Stiebel, where the left crus was extensively diseased, its diameter being more than twice that of the right, the head was always turned to the right, but there was no hemiplegia. (Vide *New Sydenham Soc. Year-Book*, 1863, pp. 90, 91; and *Medico-Chirurg. Transactions*, vol. xlvi. p. 122.

Paralysis in the form of hemiplegia is a common symptom of cerebral hemorrhage, but the amount of paralysis depends upon the damage done to the motor tract; and if this part of the brain is but little injured, there will be very little paralysis, or it may even pass entirely away. But when the corpus striatum is the seat of the hemorrhage, as is often the case, there will be marked hemiplegia, the corpus striatum, thalamus opticus, and crus cerebri being all parts of the motor tract; while if the mass of either hemisphere above the ventricles is the seat of the clot, there need be no paralysis at all. (Vide *Half-Yearly Abstract Med. Sciences*, vol. xlvii. p. 31.)

If in cases of cerebral hemorrhage the amount of the effusion be small, and it produces hemiplegia, but without any other untoward result, the patient gradually recovers. The paralysis of the face soon passes off, and the patient is soon enabled again to speak, although the paralysis of the leg and arm remain. In most cases, however, a very lengthened time is required for recovery to occur, and this is only partial or incomplete; as the clot is absorbed, the parts again come into order, and their function is resumed. But since some of the conducting fibres of the brain are absolutely severed, it is impossible for motion to be perfectly restored. As a rule, the leg recovers before the arm; but at the end of some months nearly all hope is gone of either limb permanently recovering if not restored by that time. (*Ibidem*, p. 30.)

Although cephalalgia is often observed among the premonitory symptoms of cerebral hemorrhage, it does not so often attend the extravasation itself. On this point Romberg says: Hemorrhage in the brain is much less frequently accompanied by pain than other diseases of that organ. In these cases, which may be considered as parallel to the occurrence of pain in rupture of the thoracic and abdominal viscera, the patient utters a sudden shriek; he has a distinct sense of laceration occurring in the substance of the brain. Vomiting supervenes, and the pulse becomes small and oppressed. After an interval of varying duration, the mind becomes clouded, sopor and paralysis supervene; the disease then takes its usual course, and almost always terminates in death. Cheyne describes a case of this kind (vide *Cases of Apoplexy and Lethargy*, etc., p. 110, London, 1812) which happened to a naval officer, aged 33, who, while sitting at breakfast, was seized with nausea, vomiting, and such intense headache that he said he felt that one half of the brain was torn from the other, and that his end was approaching. He rubbed his benumbed hands; complained constantly of enormous pain; was seized with rigors about midday; became comatose toward evening, and died at midnight. The post-mortem showed both hemispheres torn asunder by an enormous hemorrhagic extravasation, which had destroyed the corpus callosum and had penetrated to the base. Abercrombie communicates several illustrative cases. (Vide *Abercrombie on Diseases of the Brain*, etc., p. 218, et seq.) Headache may also occur consecutively in connection with cerebral hemorrhage, if an inflammatory condition is developed in the vicinity of the extravasation. In that case a characteristic feature presents itself in the shape of concurrent pain and contractions in the paralyzed muscles, which had previously been relaxed. (Vide *Romberg's Manual of the Nervous Diseases of Man, translated by Dr. Sieveking*, vol. i. pp. 169, 170, London, 1853.)

Sometimes the senses of hearing and sight are found to be strangely exalted in cases of cerebral hemorrhage, as they were in the following instance:

A gentleman of about forty years of age, who had a sudden attack of hemiplegia, whilst in bed heard the least sound at the bottom of the house with an acuteness which surprised him; he could also tell the hour by a watch placed on a table at such a distance from his bed as to have rendered it impossible for him to distinguish the hands when in health. (Vide *Forbes Winslow on the Obscure Diseases of the Brain*, etc., p. 447.)

The memory very often is found to be impaired in cases of cerebral hemorrhage. We append a few examples that are out of the general run:

A man, aged 65, in consequence of an attack of this disease forgot how to read, or even to distinguish one word or letter from another; but if a name or phrase were mentioned to him, he could write it immediately, and that, too, with the greatest accuracy. He was, however, incapable of reading or distinguishing what he had written; for if asked what a letter was, or how the letters were combined, it became evident that the writing had been performed *mechanically*, or without any exercise of the reflection and judgment. The means employed were not successful in restoring the knowledge of letters to his mind. (Vide *Forbes Winslow on the Obscure Diseases of the Brain*, etc., p. 267.)

"A young woman," says Dr. Shapter, "of weak intellect, subject to headache and *mal réglée*, at the age of 21 experienced an attack of apoplexy. In her convalescence it was observed that she had lost all recollection of persons and occurrences. She early recollected her mother, without the power of calling her by name; at the end of a month she pronounced some words, though but very imperfectly, and her efforts to express herself involved her in almost unintelligible periphrases."

A gentleman, after an attack of paralysis, had no recollection of the names of any of his friends. He, however, designated them correctly by mentioning their ages, with which he appeared to be familiar. (Vide *Forbes Winslow on the Obscure Diseases of the Brain*, etc., p. 266.)

Dr. Itard conceives that the loss of memory which generally accompanies apoplexy, [cerebral hemorrhage,] when it occurs in advanced life, follows in the subjoined order: there is first a forgetfulness of *proper names*, then of *substantives*, and next of *adjectives*. Adjectives appear to retain their hold with the firmest tenacity upon the mind. It is well known, says this physician, that many idiots have had a memory only for adjectives. (*Ibidem*, pp. 239, 240.)

Wepfer relates the particulars of the case of a man who, on recovering from an attack of apoplexy, was found to know nobody and remember nothing. After several weeks had elapsed, he began to notice his friends, remember words, repeat our Lord's prayer, and to read a few words of Latin rather than German, which was his native tongue. When urged to read more than a few words at a time, he said, with a heavy sigh: "I formerly understood these things, but now I do not." (Vide *Forbes Winslow on the Obscure Diseases of the Brain*, etc., p. 268, 4th ed.)

A patient whom Dr. Abercrombie attended, after recovering from an attack of apoplexy, knew his friends perfectly, but could not name them. Walking one day in the street, he met a person to whom he was very anxious to communicate something respecting a mutual friend; after various ineffectual attempts to make him understand whom he meant, he at last seized him by the arm and dragged him through several streets to the house of the gentleman of whom he was speaking, and pointed to the name-plate upon the door. A lady, after an apoplectic attack, recovered correctly her ideas of things, but could not name them. In giving directions concerning family affairs, she was quite distinct as to what she wished to have done, but could only make herself understood by going through the house and pointing to the various articles. (*Ibidem*, pp. 268, 269.)

Dr. Baillie has described a curious case of impaired memory produced by cerebral hemorrhage in vol. iv. Med. Transact. Coll. of Physicians. A gentleman, aged 56, was seized with symptoms of compression of the brain, and became completely paralyzed on the right side. It was found that he had lost the recollection of the words of his own language, except a very few which he pronounced with the greatest distinctness, and with a variety of tones to express pleasure and displeasure, joy and sorrow, to explain the circumstances of his disorder, and to give directions about what he wanted, without being aware they were not the proper words to express his meaning. (*Ibidem*, p. 273.) In the last-mentioned case, the cerebral organ of language seems to have been extensively disordered in consequence of the extravasation. The next three cases belong to the same category.

A gentleman, aged 46, who had always enjoyed good health, after experiencing great uneasiness of mind, and sustaining great bodily fatigue, was seized with apoplexy, [cerebral hemorrhage,] followed by hemiplegia. The symptoms of apoplexy were slight, but the hemiplegia was complete. The power of speech was entirely lost, so that he could only utter the sounds *ee-o*, which, however, he so varied that, with the assistance of expressive gestures, he was able to convey to those about him his meaning very distinctly upon ordinary subjects. He perfectly comprehended every thing that was said to him, and clearly understood what he meant to answer, but was able to utter only the above-mentioned sounds. Believing, however, that he actually employed the words adapted to the communication of his ideas, he often appeared surprised and displeased when he was not understood. He sometimes endeavored to explain his meaning by writing on a slate; but he generally substituted one word for another, and almost always erred in spelling what he wrote. (*Ibidem*, p. 274.)

A farmer, aged 50, was seized with hemiplegia. He never recovered the use of the affected side, and a painful hesitancy of speech also always remained. His memory was good for all words except substantives and proper names, especially the latter. This defect was accompanied with the following singular peculiarity; he perfectly recollected the initial letter of every substantive or proper name which he wished to use in conversation, though he could not recall to memory the word itself. Experience taught him the utility of preparing in manuscript a list of the things he was in the habit of calling for or speaking about, including the names of his children, servants, and acquaintances; all these he arranged alphabetically in a little pocket dictionary, which he used as follows: If he wished to ask anything about a cow, before he commenced the sentence he turned to the letter C, found the word " cow," and kept his finger and eye upon the word until he finished the sentence. He could pronounce the word cow in its proper place so long as his eyes were fixed on the written letters; but the moment he shut the book it passed out of his memory, and could not be recalled, although he recollected its initial, and could refer to it when necessary. The same obtained with other substantives and with proper names. (*Ibidem*, pp. 275, 276.)

There was at the infirmary of the Salpêtrière, according to Durand-Fardel, a woman, aged 40, and quite hemiplegic, who could only say, "*Madame été!*" . . . . "*Mon Dieu!*" . . . . "*Est-il possible?*" . . . . "*Bonjour, Madame!*" . . . . Her intelligence was perfectly preserved; she laughed at jokes which she heard, and cried when she wished to testify thankfulness for the care which was taken of her. She pronounced perfectly the few words which she could say, and these she repeated incessantly; it was, however, impossible for her to utter any thing else. (*Ibidem*, p. 375.)

In the last five or six cases, the faculty of language was singularly disordered. These patients had the disease which is now generally called *aphasia*. Hammond defines this as "a condition produced by an affection of the brain by which the idea of language, or of its expression, is impaired." (Vide *Diseases of the Nervous System*, p. 166.) It is probable that in these cases of aphasia the hemiplegia involved the right side of the body, for Seguin has shown that of 260 cases of aphasia associated with hemiplegia, the latter involved the right side in 243, and the left in only 17 instances. (*Ibidem*, p. 199.) It is also probable that in the foregoing cases of aphasia the hemorrhage or the results thereof involved the anterior lobe of the left cerebral hemisphere, for Seguin has shown that in 545 autopsies of cases of aphasia, the left anterior lobe was found diseased in 514 instances. (*Ibidem*, p. 199.) Furthermore, it is probable that, in some of these cases of aphasia, the third frontal convolution of the left hemisphere was injured by the hemorrhage, or by the consequences of the hemorrhage, for Broca and others have shown that oftentimes when aphasia is present there is some lesion of this convolution.

The following instance of cerebral hemorrhage and softening of the third anterior convolution of the left hemisphere of the brain illustrates Broca's views as to the cause of cerebral aphasia.

Mary Murphy, aged 60, was admitted to the Union Hospital, April, 1865, with right hemiplegia and defective speech. The memory of words was very defective, and the articulation confused. For "thank you, sir," she said, "fancy sell;" and, being asked what her husband (a peddler) sold, replied, "procties and pudding-pans," which was found to mean "brooches and bosom-pins." She had stenosis of the mitral orifice, and hence it was supposed that the paralysis was caused by cerebral embolism.

The right arm was rigidly flexed; the leg was weak, but allowed her to walk with a stick; the emotional faculties were very much disturbed; but the intellect was tolerably good, especially on money matters. In hospital she went by the name of "the sergeant," from the state of discipline in which she kept the patients, using her stick on occasions. Her speech could not be tested by books, for she could not read. Some words she constantly misapplied: and her attempts to talk utterly failed to convey any meaning. She was in hospital on and off for eighteen months, and, after a few days' illness, died of pneumonia.

Autopsy.—There was much subarachnoid effusion of serum; the right hemisphere was healthy, except some venous dots on the centre of Vieussens, and a little fluid in the ventricle. On carefully examining the left hemisphere, the convolution of Broca, that is, the third frontal convolution of that hemisphere, was found to be softer than the neighboring parts, and the remains of a hemorrhagic cyst, of the size of an almond, empty, but with erosion of its floor, was discovered close to the anterior third of the corpus striatum, and running parallel to its course. No embolia were found in the cerebral arteries.

The right lung was extensively hepatized, the heart fatty externally, the left auricle contained a decolorized clot moulded to its walls; buff-colored prolongations of this clot extended two inches into the pulmonary veins, and similar thrombi blocked up the right cavities and the pulmonary artery. The mitral orifice was narrow, its margins ossified; there were vegetations on the auricular surface; the aortic valves were sound. Although there was heart-disease, the hemiplegia in this case was not due to cerebral embolism, but to cerebral hemorrhage, and this fact constitutes an important feature of it. (Vide *Forbes Winslow on the Obscure Diseases of the Brain*, etc., pp. 365, 366, 4th ed. London, 1868; also, *Popham on Aphasia, or Cerebral Loss of Speech*, London, 1867.)

It will be useful to epitomize and classify the symptoms of cerebral hemorrhage.

Symptoms of effusion into the substance of the cerebral hemispheres.—Very different statements have been made as to whether premonitory symptoms are present or not, in such cases; and the practical point in diagnosis which this decrepancy of statements has taught is, "that the non-existence of precursory symptoms in a given case is in favor of the belief that hemorrhage rather than congestion is the cause of the lesion or softening."

The attack is generally *not* sudden. The symptoms are usually developed by degrees, and with varying rapidity. The correctness of this statement is proved by numerous cases from the writings of Trousseau, Abercrombie, and Mushet, which we have briefly related or referred to in the preceding pages, and by our own experience. The subject, if standing or walking while the attack is developing, may sink to earth. He falls because his leg has become paralyzed and unable to support his weight; not as if struck down by a blow, as Trousseau has clearly shown. Sometimes after a few hours the symptoms may be suddenly aggravated, in consequence of a fresh extravasation. It is the nervous symptoms, however, which have the most importance.

1. *Mental.*—Apoplectic stupor is very rarely developed all of a sudden, at the commencement of the invasion in cases of cerebral hemorrhage, unless the attack begins with epileptiform or eclampsic convulsions, and then the hemorrhage in the brain occurs as a consequence or complication of the convulsive disorder. The consciousness is generally not affected at all, or is but slightly so, at the outset. In some cases, however, the patient loses himself for a few seconds or even minutes, and then recovers his senses more or less completely. In bad cases, the patient gradually sinks into lethargy, stupor, coma, carus, and death; and sometimes these conditions succeed each other with great rapidity. But the attack is very rarely if ever ushered in with the symptoms of apoplexy, properly so-called, when the extravasation occurs in the substance of the cerebrum. Trousseau in all his vast experience never met with such a case. Not unfrequently the patient awakens after having lain in deep stupor for several hours or days. In some of these cases, the intellectual powers in time are completely restored; but in others confusion of thought, weakening of memory, and aphasia permanently remain. In all these cases, well-marked recovery of the intellectual powers affords a strong presumption that the symptoms were due to cerebral hemorrhage, and not to softening of the cerebral substance.

2. *Sensorial.*—The sensibility is, in general, less constantly and less intensely affected than the motility. When cutaneous anæsthesia is complete, although the surface so affected may be limited in extent, the occurrence indicates severity of lesion. In slight cases there is usually only numbness and tingling of the ends of the fingers. Evidence of sensation may be obtained when there is no proof of distinct volition. When profound coma is present, the dilated pupil and the half-opened eye indicate that the retina has lost its impressibility; and if the hearing and smell are similarly affected, the persistence of such symptoms are signs of evil omen. (*Aitken.*)

3. *Motorial.*—Paralysis is present in an immense majority of the cases, its characteristic form being ***hemiplegia*** ***;*** but sometimes it is general, the proportion between them being as 84 to 16 in one hundred cases; and when the paralysis is general, the hemorrhage is seldom limited to the

substance of the hemispheres. During profound stupor, the deviation of the mouth generally indicates which side is paralyzed. In less severe cases, the immobility of the limbs at the command of the will is another guide. The tongue, on protrusion, commonly deviates to the paralyzed side. Any extreme movement of the face, such as crying or laughing, renders the inequality of muscular action more apparent in the two sides. The orbicularis oculi generally escapes paralysis, or is less affected than the other facial muscles. The loss of motility is commonly absolute at first, especially in the arm, which is generally more profoundly affected than the leg, the one being more completely paralyzed than the other. Stertor is generally present after coma sets in. Involuntary contractions of the muscles of a tonic or clonic kind are extremely rare, when hemorrhage is limited to the cerebral substance. (*Aitken.*)

The more common combinations of symptoms from which the existence of cerebral hemorrhage limited to the medullary substance might be inferred are—

1. Profound coma with hemiplegia of marked intensity, and without rigidity.
2. Paralysis of both sides, but one more profoundly affected than the other—a rare occurrence in limited hemorrhage.
3. Slight coma or some other slight disturbance of the intellect, but with paralysis, hemiplegic and complete. (*Aitken.*)
4. Intellect unaffected, but with hemiplegia, the symptoms of which were gradually developed, as they were in some of Trousseau's cases.

Symptoms of hemorrhagic effusion into the ventricles.—This lesion cannot be distinguished from arachnoid extravasation in some cases, nor in others from hemorrhage into the cerebral substance, especially when it occurs in the vicinity of the ventricles. The cases, however, which are less doubtful are marked by the following characteristics, (*Aitken :*)

1. *Mental.*—The patient, after partially recovering from a slight seizure, is suddenly plunged into profound coma, from which there is no recovery. This second attack is presumed to indicate that the effusion has burst into the ventricles or into the arachnoid cavity, from its original seat in the brain-substance near the ventricles or near the convex surface.
2. *Motorial.*—Paralysis is complete in degree, and is developed simultaneously on both sides; or, after having been hemiplegia for a short time, it becomes general; when the coma of the second attack above noticed comes on, stertorous breathing is strongly marked. The pupils are dilated. Deglutition is difficult and dangerous. When the paralysis is general and the coma profound, it is almost a sure sign that hemorrhage has taken place to a considerable extent into the ventricles. (*Aitken.*)

Rigidity or tonic contraction of the muscles is present in many instances; in nineteen out of twenty-six cases it occurs in the paralyzed limbs, and in about four out of twenty-six cases may be seen in those of the healthy side. (*R. Reynolds.*) Its presence is a sign of extensive hemorrhage with laceration of the brain. The most frequent combination of symptoms indicative of hemorrhage into the ventricles may be briefly stated to be profound coma with general paralysis and rigidity. (*Aitken.*)

Symptoms of meningeal hemorrhage.—This variety of encephalic hemorrhage not unfrequently proves quickly fatal, as some of the cases which we have related clearly show. When the extravasation bursts through the pia mater and arachnoid into the cavity of the latter, the symptoms cannot be distinguished from those belonging to the analogous ventricular hemorrhage above described. If, however, the extravasation is immediately subarachnoid at first, and of limited extent, it may be approximately diagnosed: 1. By the incipient symptoms partaking the nature of those which belong to meningeal inflammation, such as severe pain in the head, with impaired intelligence, sensibility, and motility. 2. The attack is less sudden than it is when the phenomena are due to congestion, central hemorrhage, or embolism, and the symptoms are progressively developed.

The following are the combinations of symptoms which indicate the occurrence of subarachnoid hemorrhage: (1.) Complete and profound coma without paralysis, or with general paralysis very slightly developed in intensity. (2.) Complete loss of consciousness without paralysis, but combined with rigidity or clonic contractions of the limbs. (3.) Paralysis of hemiplegic distribution as regards the limbs, but without deviation of the features, the muscles of the face not being affected. (4.) Apoplectic coma without anæsthesia. (5.) Imperfectly developed coma with general paralysis. (6.) An apoplectic attack, of which the symptoms are somewhat interchangeable or intermittent. (*Aitken.*)

The *facial paralysis* which results from cerebral hemorrhage is never so complete as that which results from resolution of the seventh nerve. In the former case the orbicularis palpebrarum is never paralyzed to the same extent as it is in the latter; hence, if a hemiplegic patient be asked to shut his eye, he does it completely enough to hide the globe, while the eyeball remains uncovered in cases of paralysis of the seventh nerve. (*Trousseau.*)

Cerebral hemorrhage is attended by the group of symptoms which constitute hemiplegia more uniformly than by any others. Still hemiplegia is not characteristic of cerebral hemorrhage, for it may be produced by many other disorders. For example: Andral has related two cases, with the autopsy of each, in which hemiplegia resulted from cerebral congestion. (Vide *Clinique Médicale*, tome v. pp. 229–238, Paris, 1833.) We have related in Chapter IV. two cases belonging to the same category. Lanceraux mentions a number of cases in which hemiplegia was connected with syphilitic disease. (Vide *Treatise on Syphilis*, vol. i. pp. 193, 194, 195; vol. ii. pp. 34, 35, 55, 60, 69, 73, 79.) Trousseau refers to some instances in which it was produced by cerebral rheumatism. (Vide *Lectures on Clinical Medicine*, vol. i. pp. 521–523.) It is not unfrequently due to cerebral embolism. For a notice of four cases of this sort consult the *New Sydenham Soc. Retrospect*, 1869–70, p. 112. On this point consult also the next chapter of this book. Finally, it is sometimes produced by cerebral softening. Récamier, Trousseau, and Todd hold that whenever hemiplegia, complete and absolute, occurs *suddenly*, (and this point, that is, the suddenness of attack, is insisted on,) without loss of consciousness, softening of the brain may be diagnosed. Whenever, on the contrary, well-marked hemiplegia is attended with loss of consciousness, and especially whenever the patient has suddenly become comatose, cerebral hemorrhage may be diagnosed, and also to a considerable amount. (Vide *Trousseau's Lectures on Clinical Medicine*, vol. i. pp. 14, 15, New Syd. Soc. ed.[1])

Nevertheless, in a very large proportion of the cases where hemiplegia is present, it is due to cerebral hemorrhage, and even in those cases where it is not due to that lesion a critical examination will for the most part show to what its occurrence should really be ascribed, whether to cerebral softening, or embolism, or rheumatism, or syphilis, or congestion. Thus it appears that, although hemiplegia is not diagnostic of cerebral hemorrhage, it must be ranked among the most important of the phenomena which are produced by that disease.

Concerning the encephalitis and fever which are produced by cerebral hemorrhage.—If the patient does not die during the attack, but recovers consciousness, he shows signs of a more or less severe encephalitis in two or three days. This results from the injury of the brain-substance, produced by the extravasation, and therefore must be regarded as traumatic. When it does not reach a great height, and leads only to the formation of new connective tissue about the clot, the symptoms are, increased frequency of pulse, increased heat of skin, and other signs of fever, headache, sparks before the eyes, delirium, and occasionally twitchings and contractions or spasms of the paralyzed parts. After a time, in favorable cases, these symptoms of reaction moderate and finally disappear, and the patient is well, except the residuary hemiplegia, which is mostly motor in character. But, if the inflammation of the brain-substance in the vicinity of the clot is of great or even considerable intensity, and induces inflammatory softening, the symptoms mentioned above are accompanied by those of general paralysis, and the patient dies. In such cases, the fatal result is due to the severity of this secondary or traumatic inflammation of the brain. (*Niemeyer.*)

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[1] **Cerebral embolism, however, may, and not unfrequently does, produce exactly the same phenomena, as we shall abundantly prove in the next chapter.**

This febrile action, on which classical authors lay too little stress, rarely fails to occur when the hemorrhage is considerable. It usually commences from twenty to twenty-four hours from the outset of the attack, and reaches its maximum on the second or third day. The pulse becomes hard and frequent, the skin hot and often bathed in perspiration, the face flushed, the respiration labored, etc. The importance of this consecutive encephalitis is very great, for, much more frequently than the hemorrhage itself, the acute or inflammatory softening of the brain-substance which results from it is the cause of grave cerebral accidents, and ultimately of the patient's death. (*Trousseau.*)

Cerebral hemorrhage produces anæmia of the portion of brain-substance which is encroached upon by the extravasation. When the amount of the effusion is large, the brain-substance becomes much exsanguinated, unless it happens to be indurated and atrophied by chronic alcoholism, as was clearly shown in some cases of apoplectiform cerebral hemorrhage which we have related. Extravasation of blood in the brain suspends the cerebral functions, not because the coagulum compresses the nerve-fibres and ganglion-cells, but because it compresses the cerebral capillaries, and thus occasions anæmia of the nerve-fibres and ganglion-cells, as we have already shown in the chapter on the pathogeny of apoplexy, to which the reader is respectfully referred for an exposition of the subject. This view was first broached by Niemeyer; but other observers, among whom are Traube and Leyden, have recently adopted his conclusion.

Prognosis of Cerebral Hemorrhage.—Trousseau mentions two symptoms which possess much prognostic value in cases of hemiplegia resulting from cerebral hemorrhage. 1. Our prognosis should be unfavorable when the arm regains power more rapidly and more completely than the leg. The reverse usually obtains, and in the great majority of instances the lower limb regains the power of motion much quicker than the arm. It is remarkable, however, that when the arm recovers power quicker and better than the leg, the patient is generally much worse off than when the reverse obtains.

Dr. Ramskill has observed a case in which the patient, a gentleman between 50 and 60, after an attack of hemiplegia, soon regained complete use of his affected arm, whilst his leg remained paralyzed; but in a few months all the symptoms got worse, and in less than a year from the date of seizure, he died in a state of perfect imbecility. Trousseau relates a precisely similar case which occurred in the person of a general officer, and speaks of another case of this kind, a woman, who was then awaiting the same fate.

2. When the fingers are bent into the palm of the hand from permanent contraction of the flexor muscles, the patient will never recover the use of his hand, although the other phenomena of hemiplegia may have passed away, for the extensor muscles will never regain the power they have lost, the hand will always look like claws, and the power of motion in the upper limb will in time become almost completely abolished. We should not hold out the promise of cure, or even of improvement in such cases, because the symptoms, far from getting better, will grow worse with each succeeding year. (Vide *Lectures on Clinical Medicine*, vol. i. pp. 15–18, New Syd. Soc. ed.) This statement is perhaps too absolute, for if the contraction of the flexors is but slightly marked, and atrophy with degeneration of the extensors has not yet occurred, or, in other words, if the case be seen sufficiently early, faradization of the extensors will sometimes succeed in arresting the deformity. Dr. Bazire, the translator of this part of Trousseau's Lectures, relates a successful case of this sort.

The practitioner should be guarded in his prognosis for the first week or ten days, lest the secondary encephalitis or a fresh attack of hemorrhage should destroy the patient. Popular opinion supposes that the patient may suffer three attacks of cerebral hemorrhage, the first being mild, the second followed by hemiplegia, while the third proves fatal. It is only in a few instances that this number is exceeded. (*Aitken.*)

With regard to the hemiplegia, some improvement generally takes place even in bad cases, so that the patient recovers some use, first of his leg, and then, perhaps, of his arm, so that he is able to walk with a "straight leg" and a dragging toe or foot.

This recovery is often preceded and accompanied by severe pains, especially of the upper extremity, which indicate that the brain is still in an irritated condition. The limb, however, always wastes, and its vital powers become so much impaired that, if inflammation occur in it, it seldom ends by resolution, but has a strong tendency to gangrene, while cicatrization is slow and difficult.

The commonest occurrence is the retention or complete recovery of the mental faculty, and the progressive but much more gradual return of the sensibility and motility. Paralysis of motion sometimes persists in groups of muscles, such as those of the tongue, the forearm, or hand.

Sensibility is generally first restored, then the motion of the lower extremity, then the arm, and lastly that of the forearm and hand.

No doubt the most important of the adverse circumstances which attend recovery from cerebral hemorrhage is that, although the patient appears to be doing well for the first few days after the attack, yet toward the close of the first week the brain-substance, irritated by the clot, undergoes acute inflammatory softening, and thus induces a fatal result. Should the patient, however, survive this dangerous period, he may continue to live many months, or years, according to his age; but he is generally at length cut off by a fresh attack of hemorrhage, or his brain ultimately inflames and softens, and then he dies in convulsions or in a typhoid state.

Although it is the general rule that the patient, on recovering from the attack, has the good fortune to regain all the faculties of his mind, yet his memory is not unfrequently impaired, and sometimes to such a degree that he has forgotten all dates, the names of his friends, or even the names of things. Broussonet, professor of medicine at Montpellier, lost the remembrance of all substantives or nouns, and another case is on record in which the patient lost the recollection of all his adjectives. In some instances the faculty of association is also so much destroyed that the subject, although he remembers both names and things, is unable to connect the thing with the proper word, so that he calls that which is hot, cold, or speaks of night when he means day; or calls a coffee-pot a wash-hand basin.[1] Others, again, have forgotten how to read, and the power thus lost either returns suddenly, or they are obliged to learn *de novo.*

The ability to fix the attention is often very much impaired, and the patient is no longer able to transact business; or if he begins a sentence, is unable to finish it, or he repeats the same idea over and over again. The passions also are but little under control; for, while some weep like children, others laugh immoderately, and all are easily terrified, or otherwise easily influenced.

All these circumstances must be remembered in giving a prognosis in cases of cerebral hemorrhage. (Vide *Aitken's Science and Practice of Medicine, by Clymer*, pp. 323–325, 2d Am. ed.)

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[1] A gentleman distinguished for rank and education, and suffering from cerebral hemorrhage with aphasia, assured Romberg that, of all the inconveniences and troubles resulting from that disease, none were so painful as the fact of his employing wrong terms (such as water for food, and the like) to express his meaning.

According to Dr. Morel, the victims of cerebral hemorrhage not unfrequently lose the use of almost all the vocabulary, and only retain a knowledge of a few words which, in their estimation, have all possible kinds of meaning. When the patients are not understood, they become moody, impatient, and repeat with more or less vehemence the words they have coined. Such persons appear to have possession of their reason, which is easily known from the expression of their eyes, their gestures, and from the air of satisfaction that they show when one has guessed their meaning. This state often continues for a long time, even till death itself occurs.

TREATMENT OF CEREBRAL HEMORRHAGE.

1. *Before the attack.*—Cerebral hemorrhage sometimes occurs without warning; but when premonitory symptoms are present, the several indications which they afford should be promptly met by appropriate treatment. The cerebral hyperæmia and the increase of vascular tension, which, in most cases, are the immediate causes of these symptoms, usually demand absolute repose on the part of the patient and the exhibition of laxatives or enemata, the application of cold in the form of frozen compresses or the ice-bag to the head, and when the subject is strong and very plethoric, the abstraction of blood by venesection, or by cups, or by leeches. In sthenic cases, aconite not unfrequently proves useful. When the patient has insomnia, the bromide of potassium may generally be administered in full doses with decided benefit. If the patient has already had an attack of cerebral hemorrhage, he must be particularly careful to avoid every thing which may cause the cerebral blood-vessels to become overfilled or distended; he must especially avoid luxurious meals, indulgence in wines, beer, ale, and spirituous drinks in general, and must keep his bowels open by dieting and the use of appropriate remedies.

"The cautious abstraction of a small quanity of blood from the head when the symptoms resemble active congestion of the vessels, the administration of mercurial alteratives, a careful attention to the state of the secretions, condition of the skin, and renal functions, combined with counter-irritation and abstinence from all mental agitation and anxiety, will often be found of benefit in the incipient stages of apoplexy [cerebral hemorrhage] and paralysis. In a certain type of case connected with organic or functional disease of the heart, after relieving the local head-symptoms, great benefit will be derived from a combined use of opium, digitalis, and iodide of potassium. Where there is laborious action of the heart, consequent upon hypertrophy or valvular disease, the medicines referred to almost invariably alleviate the cardiac as well as the head-symptoms." (Vide *Forbes Winslow on the Obscure Diseases of the Brain*, etc., p. 532.)

"There are, [however,] types of incipient apoplexy and paralysis only to be successfully treated by means of tonics and stimulants. Apparently acute attacks of cerebral hemorrhage and hemiplegia yield speedily to the administration of iron, quinine, and various preparations of zinc and copper. In this anæmic class of cases, wine and generous diet will be indispensable. The pulse is generally weak, the action of the heart feeble, and the blood deficient in red globules. This is indicated by pallor of the countenance and a general state of anæmia. The patient complains of great muscular debility and nervous depression. Associated with the preceding symptoms, there will be observed *threatenings* of apoplexy and paralysis." (*Ibidem*, pp. 532, 533.)

2. *During the attack.*—The indications during this stage are to arrest the hemorrhage and to promote absorption of the extravasated blood. But we must not deceive ourselves as to our power to fulfil these indications, and must remember that we really have no remedy for stanching the hemorrhage nor for hastening the absorption of clot; and that we are practically restricted in our efforts to combating the more dangerous symptoms as well as possible, that is, to obviating the tendency to death. Venesection should generally not be employed at this stage; for it cannot stop the bleeding,[1] nor promote the removal of the effusion, and frequently will do much harm. Trousseau relates a case of cerebral hemorrhage in which the symptoms are well-marked hemiplegia, distortion of face, and impairment of speech, while the intellect was perfect. The patient was bled, but he had lost scarcely three ounces of blood when he fell into a state of collapse, from which he never rallied, and died a few days afterward. But a moment before the bleeding he was in the full enjoyment of all his faculties, and conversed freely and ably with his friends around. Trousseau, during the latter part of his life, abstained from energetic treatment in all cases of cerebral hemorrhage, in those where the symptoms were grave as well as in those where the symptoms were slight. He says, "My reasons are these: if I do not have recourse to blood-letting, purgatives, or revulsives in cerebral hemorrhage, whether considerable or not, it is because experience has taught me that the patients do better without them. And when I reflect on what happens then, I do not see how these methods of treatment can be of any use, since the hemorrhage is an accomplished fact when we are called upon to note its symptoms." . . . . "Instead of bleeding my patients, of putting them on low diet, and keeping them in bed, I do not draw blood from them, I recommend them to get up if possible, at least to remain in the sitting posture, and I feed them. I am convinced that I thus obtain much more favorable results than when I interfered more actively, and that patients so treated do a great deal better than those whom I bled in former days, kept on low diet, and confined to their beds." (Vide *Lectures on Clinical Medicine*, vol. i., pp. 9–11, New Syd. Soc. ed.)

The late Dr. Todd, in his *Clinical Lectures on Nervous Diseases*, says, "Bear in mind that in a large number of cases, probably the majority, there is in reality no cerebral congestion, and that the hemorrhage is not of a kind likely to be stopped by taking away blood. If you find your patient has been of intemperate habits, is laboring under organic disease of the heart and arteries, is of gouty or rheumatic constitution, then hesitate much before you deplete by bleeding."

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[1] Mr. Travers met with a case of cerebral hemorrhage where the attack happened while the patient was being bled for pneumonia; and numerous examples are on record of the occurrence of apoplectic stupor at the very time that bleeding was being practised for the relief of hemiplegia. (Vide *Tanner's Practice of Medicine*, p. 95, Am. ed. 1870.)

However, a case of cerebral hemorrhage does now and then occur which demands the abstraction of blood by venesection. Dr. Todd remarks: "If, upon full inquiry into all the particulars of the case, you find that your patient is of full plethoric habit, with too much blood in his body, and with a sufficiently strong heart, you may bleed him with every chance of success." The author thinks that the indications for venesection in this stage of cerebral hemorrhage may be exactly given. If the patient is purple in the face and obviously suffering from over-fulness or distention of the venous system; if, at the same time, the impulse of the heart be strong and its sounds clear; if the pulse be regular, and there are no signs of commencing œdema of the lungs, we should bleed without delay, in order to get rid of a state of general venous congestion which obstructs the flow of blood from the head, and thus promotes the tendency to death. In bad cases or feeble subjects the symptomatic indications may require us to adopt just the opposite plan of treatment, although the original disease was the same. In such cases, we must strive with all our skill to prevent paralysis of the heart by the use of stimulants, etc. If we cannot give wine, ether, musk, brandy, etc., internally, we should apply large synapisms to the epigastrium and breast, to the inside of the thighs and the calves of the legs; also rub the skin vigorously, sprinkle the breast with cold water, or drop melted sealing-wax on it, and administer stimulating enemata.

3. *After the attack.*—If the patient does not lose consciousness, or recovers it again after the attack is over, we should simply prescribe a diet consisting largely of milk and eggs or beef-tea, all articles of easy digestion, and keep the bowels open. If it is necessary to give a laxative, castor oil will generally prove to be the best. On the second or third day, when the secondary encephalitis and its accompanying fever present themselves, we should cut the hair short or shave the head and apply cold compresses or the ice-bag, according to the severity of the symptoms. It may also be advisable to administer a saline purgative, and some febrifuge medicine, such, for example, as the acetate or citrate of ammonia, but active treatment generally proves injurious. In this stage venesection is always superfluous and even dangerous. After the febrile symptoms have subsided, good may often be done by applying a blister to the nape of the neck. This epispastic may be repeated once or twice, but should generally not be continued any longer than that.

If the stage of inflammatory reaction has happily passed, and the patient is pretty well, except the paralysis, we should avoid prescribing strychnia and other remedies of a similar nature, which are neither theoretically nor practically useful, but should regulate the diet and bowels, prescribe tonics if necessary, and place the patient under the best possible hygienic influences. (*Niemeyer.*)

Lastly, it cannot be denied that paralyses are generally improved by the employment of the induced current of electricity. This is doubtless solely because "faradisation localisée" is one of the most powerful means of therapeutic gymnastics. After paralysis has lasted some time, its degree almost always depends partly on diminished excitability of the nerves, and on commencing atrophy of the muscles from long disuse. For both of these states the methodical excitement of the nerves by the induced current is certainly the best remedy, and, at all events, it deserves the preference to irritating liniments, salves, and tinctures. (*Niemeyer.*)

The case of Dr. Adam Ferguson, the historian, affords a good illustration of the benefit to be derived from prompt attention to the symptoms of cerebral plethora and hemorrhage. Dr. Ferguson experienced several attacks of temporary blindness some time before he had an attack of palsy; and he did not take these hints as readily as he should have done. He observed that while he was delivering a lecture his class and the papers before him would disappear, vanish from his sight, and reappear again in a few seconds. He was a man of full habit; at one time corpulent and very ruddy, and though by no means intemperate, he lived freely. I say he did not attend to these admonitions, and at length, in the sixtieth year of his age, he suffered a decided shock of paralysis. He recovered, however, and from that period, under the advice of his friend Dr. Black, became a strict Pythagorean in his diet, eating nothing but vegetables, and drinking only water or milk. He got rid of every paralytic symptom, became even robust and muscular for a man of his time of life, and died in full possession of his mental faculties at the advanced age of ninety-three upward of thirty years after his first attack. Sir Walter Scott describes him as having been, "long after his eightieth year one of the most striking old men it was possible to look at. His firm step and ruddy cheek contrasted agreeably and unexpectedly with his silver locks; and the dress which he usually wore, much resembling that of the Flemish peasant, gave an air of peculiarity to his whole figure. In his conversation the mixture of original with high moral feeling and extensive learning, his love of country, contempt of luxury, and especially the strong subjection of his passions and feelings to the dominion of his reason, made him, perhaps, the most striking example of the stoic philosopher which could be seen in modern days." (Vide *Sir Thomas Watson's Practice of Physic.*)

HÆMATOMA OF THE DURA MATER.

Definition.—This term signifies a flattened tumor containing blood, developed on the under surface of the dura mater.

Etiology.—This disease is always connected with chronic inflammation of the dura mater or pachymeningitis. Virchow has shown that the sacculated collections of blood occasionally found on the under surface of the dura mater, which constitute this disease, are not simple extravasations of blood at whose periphery the fibrin has been precipitated so as to incapsulate the fluid part, as was formerly supposed, but are the result of chronic inflammation of the dura mater, and hemorrhagic effusion into the newly-formed connective tissue. Virchow named this blood-sac hæmatoma of the dura mater. The blood filling it comes from the bursting of the large and thick-walled capillaries which have formed in great numbers in the pseudo-membrane of the dura mater during the course of the inflammation, and it is extravasated between the layers of this pseudo-membrane. The causes of this hemorrhagic pachymeningitis are but imperfectly known. It occurs only in the adult, and generally after the age of 50. It is met with remarkably often in drunkards, and in persons afflicted with mental disease. In some cases it appears as an independent affection, and in others as a secondary disease due to injury of the forehead. In the latter it is said that years may intervene between the injury and the first symptoms of this disorder. (*Griesinger.*)

Anatomical Appearances.—Hæmatoma of the dura mater is usually located near the sagittal suture, and has the form of an oval flattened sac, which may attain considerable size, and have a length of four or five inches, a breadth of two or three inches, and a thickness of half or three fourths of an inch. Its long diameter is parallel to the falx cerebri. The walls of the sac are colored rusty brown by altered hæmatin; its contents are partly fresh fluid or coagulated blood, partly dirty reddish-brown clots, that are unmistakably older. The corresponding hemisphere of the cerebrum is flattened, or even shows a depression. Not unfrequently the hæmatoma is found on both sides. We also, rather frequently, have an opportunity to observe the commencement of pachymeningitis hemorrhagica; for, in many autopsies, we find a delicate yellow or brown connective tissue, lodged on the inner surface of the dura mater and firmly adherent thereto.

Symptoms and Course.—Hæmatoma of the dura mater often runs its course with symptoms from which we cannot make a certain diagnosis;

and when it occurs in the course of mental disorders, we usually cannot make even a probable diagnosis. In other cases, the following factors, to which Griesinger has called attention, enable us, with more or less assurance, to make a diagnosis of hæmatoma of the dura mater. If circumscribed headache, gradually increasing to great severity, and located in the vicinity of the forehead and vertex, be the first and, for a long time, the only trouble of which the patient complains; and if, between the appearance of this pain and the occurrence of other severe brain-symptoms, there be an interval not so short as in acute diseases of the brain and its membranes, nor so long as in most chronic diseases of these parts, but especially cerebral tumors, the first suspicion falls on inflammation of the meninges, and particularly of the dura mater, since inflammation of the other membranes has so great a tendency to spread that it is accompanied by a diffuse, and not by a circumscribed form of headache. We are the more justified in holding this opinion because the form of pachymeningitis in question occurs just at the place where the pain is complained of. If the patient has been mentally diseased before the commencement of the headache, or given to excessive drinking, or if he has had an injury of the head, particularly of the forehead, some time previously, there is still more reason for supposing the case one of pachymeningitis, as is evident from the etiology. But we also know that this form of meningitis usually leads to a large effusion of blood, encroaching on the cerebral cavity, and that then the effusion is capsulated on one or both sides of the sagittal suture. Hence, if the headache be subsequently attended with signs of compression of the cerebrum, with mental disturbance, loss of memory, diminished power of thought, increased inclination to sleep which finally grows to coma, accompanied by a slowly increasing and usually not pure form of hemiplegia, after excluding various brain-diseases, we must think of hæmatoma of the dura mater as being in the front rank of those disorders which may possibly be present. Since in hæmatoma of the dura mater, absorption of the extravasated blood and consequent relief of the brain from pressure on it may occur, a favorable course of the disease and recovery of the patient speak for hæmatoma in doubtful cases. If the effusion of blood does not take place gradually, as in the above-described course of the disease, but occurs suddenly; if it is large and limited to one side, the symptoms are those of an abundant hemorrhage in the corresponding hemisphere of the cerebrum. On superficial examination, it may appear remarkable that occasionally, even in large hæmatomata limited to one side, there is no hemiplegia at all, or it is very incomplete; but we must bear in mind that hæmatoma occurs just at the place where the increased pressure on one hemisphere is most readily transmitted to the other, through the free communication between the two sides which exists in the anterior portion of the skull, particularly when the hemorrhage comes on slowly. Among the symptoms of hæmatoma, Griesinger also lays particular stress on the almost constant contraction of the pupil, and is inclined to regard this as a "symptom of irritation of the surface." (*Niemeyer.*)

But this disease, after long-continued cephalic misery, generally terminates in sudden death with apoplectiform phenomena.

Treatment of Hæmatoma of the Dura Mater.—The therapeutics of this disease are not settled. Dr. Aitken thinks that the treatment should be more or less expectant. The newly-formed membranes tend to undergo retrograde metamorphosis, and thus finally disappear. To promote this should be the object of any rational plan of treatment, which must be based upon the special history of the individual case, especially as to the previous existence of syphilis or not.

Niemeyer says, if we consider our diagnosis of hæmatoma in a recent case as certain, we may apply leeches behind the ears, ice-compresses to the head, and occasionally give a purge. In the advanced or late stages, blisters or pustulating ointments applied to the nape of the neck suffice. He also informs us that with this plan of treatment he has obtained very good results in two cases; but admits that, although the symptoms in these cases were quite characteristic, there may, possibly, have been an error in diagnosis. (Vide *Text-Book of Practical Medicine*, vol. ii. p. 205, 1st Am. ed.)

Opinions of other Writers concerning Hæmatoma of the Dura Mater—Charcot and Vulpian give their adhesion to Virchow's views as to the prior existence of inflammation of the dura mater, and the formation of vascularized false-membrane, from which the hemorrhage proceeds. They also agree with Schuberg as to the existence of prodromata, which may extend over several months, and consist in general weakening of the memory and of the intelligence, giddiness, and continuous or intermittent, general or local pain in the head, followed at a later period by aggravation of these phenomena, transitory loss of consciousness from momentally arrested cerebral circulation, somnolence and apathy, weakness, and generally one-sided paralysis of the extremities, which soon disappear. Finally, the apoplectiform phenomena occur, which, of course, vary according to the magnitude of the extravasation. They believe that the newly-formed membranes may undergo retrograde change, and even quite disappear, and think that to effect this should be the object of treatment in suspected cases of pachymeningitis hemorrhagica. (Vide *New Sydenham Soc. Year-Book*, 1862, pp. 77, 78.)

Lancereaux also believes that the pseudo-membrane is first formed, and that the hemorrhage is secondary, in cases of hæmatoma of the dura mater. The seat of the pseudo-membranes, according to this observer, is almost always the convexity of the cerebrum, on one or both sides, usually in the vicinity of the division of the middle meningeal artery; here also the layer is thickest, and here also the sanguineous effusions are usually formed. The pia mater, as well as the dura, commonly presents marks of hyperæmia or of inflammation, and even of suppuration. The symptoms vary according to the age and the development of the pseudo-membranes, and the gradual or sudden effusion of bloody or serous fluid. If the membrane exists alone, its presence is indicated by pain only, limited usually to the seat it occupies; and, perhaps, by giddiness also. When hemorrhage has taken place, there are signs of pressure on the brain, such as dulness, somnolence and paralysis, and of excitement, as manifested by contraction of the pupils, and of the extremities, and sometimes by convulsions. The transitory and interchanging character of the symptoms is also characteristic. The skin is usually pale and cachectic, the pulse normal, emaciation occurs, and sometimes vomiting. The sudden attacks of hemorrhage which subsequently take place do not essentially differ from the primary extravasation. They induce the occurrence of apoplectic or convulsive attacks with secondary paralysis or contractions, sometimes delirium, but for the most part general paralysis and coma. These phenomena occur suddenly after a varying duration of the general symptoms, especially of pain in the head, and usually prove speedily fatal. The course is generally chronic; in rare cases it may end in recovery, the symptoms at all events disappearing, although the morbid formation remains. The chief causes are injuries of the skull, the abuse of alcoholic drinks, rheumatism, and erysipelas of the scalp. (Vide *New Sydenham Soc. Year-Book*, 1863, p. 88.)

All agree that this disease is partly inflammatory and partly hemorrhagic in its origin, that the inflammation occurs in the dura mater, and the bleeding from the young capillaries which have been developed in the pseudo-membranous products of the inflammation.

CHAPTER VI.

ON CEREBRAL EMBOLISM, ESPECIALLY APOPLECTIFORM CEREBRAL EMBOLISM, OR EMBOLIC APOPLEXY: ALSO ON THROMBOSIS OF THE CEREBRAL ARTERIES.

Definition of embolism.—Dr. Kirkes's paper on embolism.—Dr. Kirkes's three cases, and many other instances of it mentioned.—Three cases from Trousseau briefly related. —*Case* XXXIII. Illness, with obscure symptoms, of five weeks' duration; then coma and hemiplegia, with clonic spasms of right side, suddenly occurred; death thirty-six hours afterward; autopsy; mitral valve of heart diseased, with so-called vegetations and coagula thereon; renal and splenic embolism; cerebral embolism doubtless also present.—*Case* XXXIV. Several attacks of articular rheumatism; symptoms of cardiac disease present; symptoms also of embolism in the arteries of both the upper and lower extremity on the left side appear; death; autopsy; vegetations on mitral and aortic valves; middle meningeal, (left,) left axillary, the mesenteric and splenic arteries, and the abdominal aorta obstructed with embolia; yellow infarctions in kidneys and spleen, etc.—*Case* XXXV. Apoplexy and hemiplegia of right side, occurring in connection with acute pleurisy; death two and a half months afterward; autopsy; embolism of left arteria fossæ Sylvii; cerebral softening, etc.—*Case* XXXVI. Pleurisy attended with apoplexy and hemiplegia of left side; death three months afterward from exhaustion; autopsy; embolism of basilar artery; right cerebral hemisphere extensively softened, etc. Pleurisy may terminate in sudden death by inducing cerebral embolism. The subject of great practical importance. Pneumonia also may give rise to cerebral embolism.—*Case* XXXVII. Pneumonia; convalescence; sudden seizure with hemiplegia, pallor, pulselessness, and gasping respiration; death occurred in fifteen minutes; autopsy; embolia found in middle cerebral and coronary (of heart) arteries; clot in ascending aorta, etc. Gangrene of the lungs sometimes induces cerebral embolism. Phthisis pulmonalis occasionally produces cerebral embolism; several instances mentioned. Remarkable case by Dr. Markham in which apoplexy, hemiplegia, and death in sixty hours were produced by embolism of the great arteries of the brain and neck. —*Case* XXXVIII. Melancholia from the occlusion of numerous minute arteries of the brain with embolia derived from the mitral valve of heart; also commencing hæmatoma of the dura mater; it was an instance of capillary embolism of the brain.—*Etiology of Cerebral Embolism;* it generally results from valvular disease of the heart; sometimes, however, it is induced by pleurisy, pneumonia, pulmonary gangrene, pulmonary consumption, the bursting of the atheromatous, fibrinous, or purulent cysts of arteries into their calibre, and by the detachment of coagula from aneurisms of the neck, as in Esmarch's case; ulcerous endocarditis; capillary embolia; pigment embolia; cerebral embolism most frequent before middle age; author's statistics.—*Anatomical Appearances produced by Cerebral Embolism;* anæmia of brain-substance; passive hyperæmia of brain-substance; Prevost and Cotard's experiments; the reason of the difference; the hyperæmia not inflammatory; cerebral softening or necrosis; its most frequent seat; its color may be *red*, *yellow*, or *white;* characteristics of brain-softening from cerebral embolism; brain-softening may be produced by encephalitis—its characteristics; by constitutional syphilis and by mechanical pressure from tumors, etc.; embolism of a cerebral artery not unfrequently accompanied by thrombosis of the same artery; but thrombosis of the cerebral arteries sometimes occurs independently of embolism, for example, in the aged as a result of chronic endarteritis or atheromatous degeneration, in the young as a consequence of acute arteritis of a local or circumscribed character.—*Case* XXXIX. Hemiplegia of right side with temporary loss of consciousness suddenly occurred without apparent cause; death forty-eight days afterward with symptoms of cerebral softening; autopsy; middle cerebral artery on each side inflamed and occluded by a thrombus; left corpus striatum softened and discolored, etc.—*Cases* XL. and XLI. In both death suddenly occurred with symptoms of intense apoplexy; thrombosis of the basilar artery resulting from inflammation of the walls of that artery was found in both; the inflammatory changes in the arterial coats minutely described; the phenomena of apoplexy, properly so-called, may be produced by thrombosis of the cerebral arteries. Finally, the plugging of a cerebral artery in consequence of embolism, or thrombosis, or both combined, occasionally gives rise to cerebral or intracranial aneurism; an instance related.—*Symptoms and Course of Cerebral Embolism*; suspension of the cerebral functions in consequence of arrest in the blood-supply to a portion of the brain; hemiplegia, loss of consciousness, and apoplectic coma; examination of heart and lungs in doubtful cases; symptoms of recovery.—*Case* XLII. Chronic valvular disease of heart; hemiplegia of left side and apoplectic coma suddenly occurred; partial recovery; death about three months afterward from cardiac and renal dropsy; no autopsy; had previously suffered an admonitory embolism of the left brachial artery.—Symptoms of embolism on proximal side of circle of Willis; do. on distal side.—Recovery possible only when the collateral circulation is established in less than forty-eight hours.—Symptoms of pigmentary embolism of the brain.—*Diagnosis of Cerebral Embolism.* 1. The symptoms of hemiplegia and apoplectic stupor when due to cerebral embolism are almost always suddenly developed at the commencement of the attack. 2. Cerebral embolism is always preceded by characteristic premonitory symptoms; they are enumerated. 3. The age of the patient may be an item of some importance.—Pigmentary embolism of the cerebral capillaries occurs only in cases of long-continued malarial intoxication.—*Treatment of Cerebral Embolism;* we cannot remove the obstructing coagula with therapeutic agents; the indications are to favor the development of a collateral circulation without exposing the patient to fresh dangers; we should be very careful about employing venesection in these cases; tonics and nourishing food required much oftener than sedatives and a spare diet; preventive measures that may be employed in cases predisposed to the occurrence of cerebral embolism.

Definition.—The term embolism is derived from the Greek word ἔμβολος, *a plug*, and signifies that some blood-vessel, usually an artery, is plugged up with a clot of blood, or a concretion of fibrin, or a fragment of broken-down tissue, which has been brought by the circulating blood from some distant part. The term cerebral embolism denotes that some artery or arteries of the brain have become obstructed in this way, and the term embolic apoplexy, or apoplectiform cerebral embolism, is applied to the cases in which embolism of the arteries of the brain is attended with phenomena of apoplexy properly so called.

In the transactions of the Medico-Chirurgical Society of London for 1852, (at pp. 281–324,) was published a singularly valuable paper, by Dr. William Senhouse Kirkes, "*On some of the Principal Effects resulting from Detachment of Fibrinous Deposits from the Interior of the Heart,*" etc.,[1] wherein he presents, among some others, the following conclusions: 1. That fibrinous concretions on the valves or interior of the heart admit of being readily detached during life, and mingled with or carried away in the circulating blood; 2. That when detached and borne away in large masses, they may suddenly block up some large artery, and thus cut off the supply of blood to some important part; when in smaller masses, they may be arrested in arteries of a much smaller size, and give rise to various morbid appearances in the affected organs, (p. 324.) In this way embolism of almost any artery of the whole body may be produced. Embolism of the middle cerebral artery occurred in the first three cases related by Dr. Kirkes. The following is a brief abstract of them:—

1. A woman, aged 34, suddenly became paralyzed on the left side as to motion, and speechless, but without loss of consciousness; sensation unimpaired; death occurred at the end of five days.

Autopsy.—The right middle cerebral artery just at its commencement was plugged up by a small nodule of firm, whitish, fibrinous-looking substance; right corpus striatum much softened; right cerebral hemisphere generally contained less than the normal quantity of blood, and was softer than natural in consistence. The mitral valve was much diseased, the auricular surface of its large cusp being beset with large warty excrescences of adherent blood-stained fibrin. The right common iliac artery was also obstructed. Death was due to softening of the brain.

2. A woman, aged 24, had hemiplegia of right side, which came on suddenly; complete loss of motion; partial loss of sensation; consciousness unimpaired; died three months after attack. On post-mortem examination, the left middle cerebral artery, immediately after its origin, was found plugged up by a firm, whitish, oval mass, about the size and shape of a grain of wheat; left cerebral hemisphere extensively softened. The embolus came from the mitral valve.

3. A gas-fitter, aged 24, of intemperate habits, suddenly became hemiplegic on his left side; motion completely abolished; sensation not much impaired; did not lose consciousness; death occurred twenty-four days afterward.

Autopsy.—The right middle cerebral artery was occluded just at its origin by a firm plug of pale fibrinous substance; vessels of pia mater unusually deficient in blood, almost empty; substance of brain remarkably pale, soft, and watery in every part. The mitral, aortic, and tricuspid valves were encrusted over with large, firm, warty granulations. In each of these cases the cerebral embolism produced cerebral anæmia and cerebral necrobiosis or softening. In each of them the embolus consisted of a concretion of fibrin which had migrated from the valves of the heart. In each of them, also, the symptoms consisted essentially in the sudden occurrence of hemiplegia, but without loss of consciousness. The cerebral anæmia does not seem to have been extensive enough for that. These, then, were not cases of apoplexy, but still they serve a good purpose for illustrating many points in the history of cerebral embolism.

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[1] The first rational explanation of embolism was, however, made by Virchow, in 1847, who, in a paper on acute inflammation of the arteries, distinctly explained the manner in which the vessels were occluded by clots transported in the blood from distant parts of the body, and who associated these coagula with valvular disease of the heart. In two of the cases cited by him in which arteries were found closed by such clots, the valves of the heart were discovered to have others still attached to them, and exhibited traces of the separation of those which were found in the vessels. (Vide *Archiv für Pathol. Anat.* B. 1. 1847, p. 272; also *Hammond's Diseases of the Nervous System*, p. 134.) But Dr. Kirkes appears not to have been aware of Virchow's prior researches. Moreover, as stated above, his paper is of remarkable value, as an exposition of the subject.

Since the publication of Dr. Kirkes's paper many cases of cerebral embolism have been put on record. For example, Jenner has reported a case of embolism of the cerebral arteries, the result of rheumatic endocarditis, which produced hemiplegia. (Vide *Lancet*, May 12th, and July 14th, 1860.) Lebert has related a very interesting instance: "Symptoms of endocarditis with insufficiency of aortic and mitral valves; apoplectic attack; pneumonia; death; double embolism of the arteria fossæ Sylvii with corresponding softening of the brain; valves on left side of heart diseased." (Vide *Edinburgh Med. Journal*, Aug. 1860, p. 169.) Anstie has recorded a case of hemiplegia resulting from embolism. (Vide *Patholog. Transact.*, vol. x. p. 44.) Joffroy has given a case which occurred in a female, aged 34, of embolism of the right arteria fissuræ Sylvii, associated with considerable narrowing of the mitral orifice; the embolus became organized and contracted, so that one part of the artery became able to transmit blood again. Death occurred four months after the apoplectic attack. (Vide *Gazette Méd. de Paris*, 1869, p. 446.) Lionville has recorded a case of sudden death occurring in a woman of 78, who had incomplete left hemiplegia; the autopsy showed ischæmia of nearly the whole right cerebral lobe, and embolism involving the right internal carotid at its termination, and its two branches; the embolus was hard, well-formed, rounded, smooth, and not adherent. (Vide *op. cit.* p. 519.) Orr has furnished a case of advanced disease of the aortic valves in a boy of 13, with embolism of the left artery of the Sylvian fissure. (Vide *Glasgow Med. Journal*, 1869, p. 145.) Cases of cerebral embolism have been published by Oppolzer, Addison, Rees, Jenner, Frames, Thudichum, Walthen, Tüngel, Bristowe, Ogle, Buhl, Luton, Kapff, Lancereaux, and others. (Vide *New Sydenham Soc. Year-Book*, 1861, p. 180; also *do. Retrospect*, 1869–70, pp. 152–155.)

Trousseau has related the following cases in his Lectures on Clinical Medicine:

1. "Some years ago, a young married lady, whom I was seeing in

consultation with my friend, Dr. Voillemier, felt uncomfortable sensations in the region of the heart, and was afterward suddenly seized with painful tingling in the fingers. They had a bluish color, and soon presented all the appearances of a dry gangrenous affection; fortunately, the gangrene was limited to one of the last phalanges, which the patient lost. Eighteen months later, this lady was suddenly struck with complete paralysis of one side of the body, and subsequently sunk with all the symptoms of softening of the brain. The age of the patient made it improbable that she had had an attack of simple apoplexy; the suddenness of the paralytic seizure and the nature of the symptoms which had eighteen months previously shown themselves in the hand led me to conclude that on both occasions something similar had occurred. On the first occasion, an embolium having obliterated an artery of the hand, caused gangrene of a finger; and on the second occasion, an embolium penetrated some artery of the brain, and produced softening, a sort of cerebral gangrene, of which hemiplegia was the principal manifestation."

2. "Nearly at the same time, a friend, quite a young man, was carried off by an attack of paralysis, which supervened under circumstances similar to those just described; the hemiplegia with which he was suddenly seized came on abruptly in the course of an attack of rheumatism of the heart."

3. "A woman, aged 47, admitted to hospital for valvular disease of the heart, anasarca, and ascites, had, after some time, experienced much benefit; the anasarca had disappeared and the ascites decreased; when she suddenly felt very acute pain in the right side of the head, and was struck with hemiplegia on the left side of the body. She retained consciousness up to death, which ere long occurred."

Autopsy.—"The middle meningeal artery, (of the right side,) which presented no trace of ossification, was completely obliterated by a clot of blood, black, homogeneous, and three centimetres in length; the branches going to the right corpus striatum, which was in process of softening, were also obliterated in the same way. All the other arteries were free from obstruction." There were "also serious lesions at both auriculo-ventricular orifices, the valves of which were indurated, adherent, and insufficient. There also existed dilatation with hypertrophy of the heart."

4. A woman recently delivered, was admitted to hospital in an unconscious state, with complete hemiplegia as to motor power on the right side, but no diminution of the sentient power. She had been struck with paralysis on the previous evening, but without immediate loss of consciousness, as she was able to say, "Take me to the hospital." Her pulse was frequent and irregular. Auscultation of the heart showed a bellows-murmur, having

its maximum intensity at the apex, beyond the nipple. This murmur was harsh, and the mitral orifice was its probable seat. Trousseau diagnosed embolism of the middle cerebral artery of the left side. The patient died eight days after her admission to hospital, without exhibiting any inflammatory reaction, without regaining consciousness, and without showing any change in the state of the paralyzed side.

Autopsy.—A portion of brain in front of the left corpus striatum was softened; on the same side, the calibre of the middle cerebral artery, at the place where it expands into a dense vascular network, was obliterated to the extent of two millimetres, by a small fibrinous clot which was yellow, resistant, and did not adhere to the walls of the vessel; all around this little fibrinous clot there was coagulated blood in the artery, which, on the one side, became lost in the anastomosing branches of the vessel, and, on the other, terminated abruptly at the origin of the artery. The little central clot which thus plugged the middle cerebral artery was like a millet-seed. There was no lesion of the walls of the vessel. The plugging or embolism had been followed by thrombosis.

At the bifurcation of the left common carotid artery, there was also a small fibrinous clot having the size of a millet-seed, which sent three filiform prolongations, cruoric and fibrinous, one into the common carotid, another into the internal carotid, and a third into the middle thyroid.

The mitral valve was the seat of conspicuous lesions. On the auricular surface of the valve were warty-looking clots of various sizes, some adherent to the endocardium, and others so free as to be almost unattached. Now, it would be difficult to avoid seeing that there was a relation of cause and effect between the mitral lesion and the embolism of the middle cerebral artery, together with the softening of the cerebral substance which accompanied it. Observe also that the other arteries of the brain, examined with care, presented no intra-vascular coagula, and that, except in the situation of the softening, the entire brain was normal in color and consistence. Finally, the similarity in form and the identity in structure of the fibrinous concretions on the mitral valve with that which was found in the middle cerebral artery afford an additional reason in favor of the view that it had migrated from the heart. (Vide *Trousseau's Lectures on Clinical Medicine*, vol. iii. pp. 414–418. New Sydenham Soc. ed.) Trousseau, on examining this case for the first time, inferred that embolism of the middle cerebral artery on the left side had taken place, from the presence of hemiplegia on the right side, from the existence of some considerable lesion involving the mitral valve of the heart, from the facility with which sanguineous coagula are known to form in recently delivered women, and from the suddenness of the attack or the rapidity with which the symptoms were developed. And, generally, the coincidence of an organic lesion involving the valves of the left side of the heart with the symptoms of apoplexy and hemiplegia, leads us to infer that cerebral embolism has taken place.

CASE XXXIII.

Illness with obscure symptoms of five weeks' duration; coma, and hemiplegia with clonic spasms of right side then suddenly occurred; death about thirty-six hours afterward; autopsy; mitral disease of heart, with so-called vegetations and coagula thereon; renal and splenic embolism; cerebral embolism also doubtless present. This case was reported to the Pathological Society of Philadelphia by DR. HUTCHINSON. (Vide *American Journal of the Medical Sciences* for October, 1866, pp. 420–422.)

Joseph Hall, aged 30, formerly a soldier in the British army, was admitted to the Episcopal Hospital, March 2d, 1866. The symptoms were not marked. He had slight hebetude, a coated but moist tongue, a pale face, slight headache, one or two spots resembling slightly the rose-colored *taches*, and disappearing under pressure, but no tympanitis, abdominal tenderness, gurgling, diarrhœa, nor râles in the chest—he had, however, been subject to epistaxis. The previous history, obtained from himself, was not satisfactory. His sickness had lasted four weeks. It came on suddenly with vomiting and intense pain in the small of the back, followed by painful cramps in the leg and some difficulty in passing water. Pressure over the kidneys caused pain, especially on the right side.

His urine contained a small quantity of albumen, epithelium from the bladder and pelvis of the kidney, and some crystals of the triple phosphate. There was no dropsy. No change in his condition occurred until March 8th. Up to that time he was treated as a mild case of typhoid fever. On the afternoon of that day he was taken with nausea and vomiting, and cramps in the leg occurred for the first time since his admission. In the evening his symptoms were entirely relieved.

March 9th. Dr. Watson found the patient comatose, at his morning visit, and was told that he had been so for five hours. The pupils were widely dilated and insensible to light; the respiration stertorous; pulse 100; furthermore, there was paralysis of the whole right side, (hemiplegia,) which was also subject to clonic spasms, and to the hand appeared very much less warm than the other side, although this was not confirmed by the thermometer. Dr. Watson believed the case to be one of uræmic poisoning; he applied cups to the loins, and administered an enema containing Epsom salts and assafœtida.

"At 10 A.M.," says Dr. Hutchinson, "I saw the patient, and then for the first time heard a rough, rasping murmur at the apex of the heart, synchronous with the systole, and communicating a sense of fremitus to the præcordial region. I directed a blister to be applied to the back of the neck, and a free evacuation to be procured from the bowels. My own diagnosis of the case was embolism of one of the cerebral arteries, and I was led to form that opinion by the grave cerebral symptoms present—the comatose condition, hemiplegia, and stertorous respiration; the age and appearance of the patient almost excluding the idea of apoplexy. I thought there was no sufficient reason to entertain the view of uræmia—there had been no suppression of urine, and the quantity of albumen had always been small."

March 10*th.* No motion having occurred, croton oil was administered, and produced a large evacuation. The pulse being full and tense, blood was drawn from the temporal artery to the extent of five fluid ounces. Patient died at 6 P.M.

Autopsy eighteen hours after death.—While removing the brain, the arteries at its base were torn, and no embolus could be found. The membranes were slightly congested; the ventricles contained a small amount of serum; the brain-substance was carefully examined, but nothing abnormal was discovered.

The mitral valve of the heart was insufficient, and covered with vegetations about which the fibrin of the blood had coagulated; the other valves were healthy. The lungs were healthy except that the right one contained some distinctly circumscribed congested points at its apex and back part of upper lobe.

The liver was healthy. The kidneys were normal in size, but their section exhibited pyramidal spaces where the color of the renal substance had changed to a brownish-yellow; these were found in by far greater number in the right kidney. The same condition existed in the spleen. Some of the spots evidently were of more recent origin than others.

Careful dissection revealed a clot in the branch of the renal artery which supplied the largest of the patches described above.

The post-mortem urine was examined, but, although it was found to contain albumen in somewhat greater quantity than before, and epithelial cells in abundance, no distinctly-marked tube-casts were seen.

A microscopical examination of the kidneys and spleen showed the disordered tissue to be in a condition of retrograde metamorphosis—the cells of the kidneys being very granular and containing some oil; besides which a large quantity of free oil and granules was found in the field of the microscope.

Dr. Hutchinson remarks that, although no clot was found in the cerebral arteries, he feels convinced that a more careful examination would have revealed the presence of a cerebral embolus. The morbid state of the kidneys and spleen was unmistakably due to plugging of some of their smaller arteries; and no other hypothesis but that of cerebral embolism serves to account satisfactorily for the head-symptoms—the sudden occurrence of hemiplegia, coma, and death; for there was no cerebral hemorrhage; and the amount of cerebral congestion and cerebral œdema was not sufficient to have caused them. Moreover, this man had an affection of the heart which is especially prone to occasion embolism in distant parts through the migration of vegetations and clots from the mitral valve, they being carried away in the current of circulating blood, and the cerebral symptoms which he exhibited were precisely the same as have been observed in cases where the middle cerebral artery on the left side was found plugged up with embolia on making a post-mortem examination.

The intense pain in the loins, of which the patient complained at the beginning of his sickness, was probably connected with the occurrence of renal embolism, that is, it was coincident with and resulted from it. When embolism occurs in the extremities, it not unfrequently gives rise to pain of a most excruciating character. The albuminuria and the other signs of renal disease which he exhibited were probably due to the infarctions of the kidneys which the plugging up of the renal arteries induced; and the obscure symptoms of which he complained, both before and after admission to hospital, were for the most part occasioned either directly or indirectly by the renal embolism. This case is, therefore, a most instructive one to the student of internal pathology. In the end it was found that the disease for which this man entered the hospital was an organic lesion of the mitral valve of the heart, and infarctions of the kidneys resulting from occlusion of some of the branches of the renal arteries with embolia which had been washed off from the diseased valve by the arterial stream, and there is no good reason to doubt that the disorder which suddenly deprived him of consciousness, paralyzed the right side of his body, and in a short time afterward destroyed his life, was plugging up of the cerebral arteries with embolia derived from the same source.

CASE XXXIV.

Several attacks of articular rheumatism; signs of cardiac disease; signs also of embolism in the arteries of both the upper and lower extremity on the left side; death; autopsy; vegetations on mitral and aortic valves; left middle meningeal, left axillary, the mesenteric and splenic ar-

teries, and the abdominal aorta obstructed with embolia ; yellow infarctions in kidneys and spleen, etc.

Dr. Loomis exhibited to the New-York Pathological Society, November 23d, 1870, a specimen taken from a German woman, aged 32, who was admitted to Bellevue Hospital, Nov. 10th. Nine years ago, she had her first attack of rheumatism, three years ago her second attack, and a year ago her third attack. Each of them confined her to bed for two or three weeks. Except these attacks, she reported herself as perfectly healthy up to last May, when she began to complain of pain in her left side and shortness of breath on exertion. She also had cough with rather tenacious sputa. Last August, she was confined, at full term, with a healthy child. After that she failed rapidly. When admitted to hospital, her temperature was 102°, her pulse 100, her respiration 30, and she was very much emaciated; she located the pain in her left side in the cardiac region, and her expectoration somewhat resembled that of pneumonia.

On physical examination there was slight dulness on percussion over the posterior portion of both lungs, with diffused bronchial respiration, which was most marked on the left side. There was also an increase of vocal resonance, but no râles were appreciated at the time. The apex of the heart beat to the left of the nipple, the cardiac impulse in general was abnormally diffused, and accompanied by a purring thrill. At the junction of the third rib, and just behind the sternum, a pulsation synchronous with the cardiac impulse was distinctly felt. On percussion the area of the heart was found to be increased. On auscultation a distinct murmur was heard, at the junction of the third rib with the sternum, during and after the second sound, having its maximum intensity at that point, and being also propagated along the sternum down to the xiphoid cartilage. Another murmur was heard before the first sound, and with greatest intensity at the apex. It was of a blubbering character, and imparted to Dr. Loomis the idea of vegetations. There was still another murmur which followed the first sound, and with it was heard with greatest intensity at the apex, conveyed also to the left, and heard behind the angle of the scapula. Dr. Loomis also thought that he detected a fourth murmur at the base. From this time pulsation in the carotids was observed, and she very steadily lost flesh. It should be mentioned that there was a node on her forehead and several on her tibiæ.

November 15th, five days after admission, it was observed that the radial pulse was very indistinctly felt in her left arm, and that the artery beat indistinctly until the axilla was reached. At the same time it was noticed that pulsation had ceased in the left femoral artery, and in the common iliac below the bifurcation.

November 18th, she had a severe attack of dyspnœa. The house-physician examined her at the time and discovered crepitant râles over the posterior part of both lungs. These râles, and others of a smaller size, were heard in a circumscribed area. On the following day they had almost entirely disappeared, giving place to the same diffused bronchial respiration which was heard when the patient came to hospital.

November 20th, she became delirious. A complete loss of the radial impulse on the left side was then observed; in fact, no pulsation could be discerned below the axilla on that side. The pulsations in the arteries of the right side were good. Gangrene had commenced in the left foot as a result of arterial obstruction. The temperature of the two sides differed materially in consequence of the differences in circulation. In the right axilla it was 102¾°; in the left, 100°; in the right inguinal region, 101°; and in the left, 97°. This difference in the temperature lasted until death. The delirium continued, she passed into a typhoid condition, the heart's action became irregular and intermitting, she became unconscious on the evening of the 20th, and sank.

Autopsy.—On opening the cranial cavity, about four ounces of fluid were found in the arachnoid. The arachnoid itself did not present any abnormity. The left middle meningeal artery was found to be plugged. There were no points of softening in the brain-substance. The pericardium was found attached to the pleura, and the two surfaces were agglutinated to each other. The pericardial sac contained about twelve ounces of fluid. There were firm bands of adhesions between the two surfaces of the pericardium, and these bands were of considerable length at certain points. The right side of the heart was normal; the left ventricle, however, was very much dilated, and its walls hypertrophied. The mitral and tricuspid valves were thickened, and their edges at different points seemed to be adherent. On the ventricular surface of the aortic valves was a vegetation, having the size of a pea, and standing out very prominently. The mitral valve exhibited vegetations on both surfaces. The axillary artery on the left side was plugged up with fibrin, as was also the abdominal aorta about two inches above its bifurcation. This plug extended into the femoral, but only partially obstructed that vessel. The superior mesenteric artery also was plugged up from its origin for an inch and a half.

The lungs were firmly adherent throughout, and the cavity of the left chest contained considerable serum. The right lung was œdematous. There were no traces of pneumonia in either lung. There were yellow infarctions in the kidneys and spleen, and one of the arteries of the latter organ was plugged. (Vide *The* (N. Y.) *Medical Record*, vol. v. pp. 543, 544.)

Comments.—The embolism in this case was very extensive, for it involved the femoral, the iliac, the abdominal aorta, the superior mesenteric, the splenic, the left axillary, and the left middle meningeal artery. The embolia were formed in the left heart in consequence of rheumatic endocarditis, and were swept away from it by the current of the circulating blood. So far as the head was concerned, the embolism was meningeal rather than cerebral. The delirium and the typhoid state into which she sank and died were probably due to the septæmia that the gangrene of her foot, etc., had induced; and the gangrene itself was occasioned by embolism of the lower extremities.

But cerebral embolism may be induced by certain diseases which do not involve the structures belonging to the heart. We shall now proceed to relate some, and to refer to other cases in which this affection of the arteries of the brain was due to pleurisy or perhaps pleuro-pneumonia, to pneumonia, and to phthisis pulmonalis.

CASE XXXV.

Apoplexy and hemiplegia of right side, occurring in connection with acute pleurisy; death two and a half months afterwards; embolism of left arteria fossæ Sylvii; cerebral softening, etc.

M. VALLIN reports the case of a soldier, aged 21, who was admitted to Val-de-Grace with pleurisy on the left side and effusion filling about two thirds of the pleural cavity. Heart pushed over to the right side. No abnormal bruits. Right side of chest healthy. In spite of an energetic treatment, the effusion increased and the fever persisted. Thirteen days after admission, the patient suddenly became unconscious and hemiplegic on the right side, the sensibility remaining intact. Thoracentesis, already indicated, was immediately performed, but with no good result, and was repeated at the end of a week. At this time a loose patch of gangrene was noticed on the sole of the right foot. The symptoms abated after the second operation, and the paralysis gradually disappeared. The pleuritic effusion, however, returned and necessitated a third performance of thoracentesis. The patient slowly sank from exhaustion, and died at the end of the third month after admission.

At the *autopsy*, a patch of softening was found in the left cerebral hemisphere, near the corpus striatum; and, at the posterior extremity of this patch, an excavation filled with a creamy fluid, the walls of which were irregular in shape, bright yellow in color, and composed of soft and pulpy cerebral tissue retained in a network of fine and pale capillaries. This pulpy material presented under the microscope capillary loops, abundant fatty molecules, granular corpuscles analogous to those of colostrum, some hematoidin, and some altered blood-corpuscles.

Two branches of the left arteria fossæ Sylvii were found completely obliterated by a fibrinous clot.

The heart and large vessels were perfectly healthy, and in no part of them could a trace of clot be found; but more than two and a half months had elapsed since the apoplectic symptoms occurred. It is probable that slight and temporary thrombosis of the pulmonary veins, or of the left side of the heart, produced some clots which, by migrating, plugged up the left Sylvian artery or several of its more important branches. In such a transient thrombosis with migration of the clots we may find the explanation of certain sudden deaths in cases of pleurisy, occurring either during the course of the effusion or after thoracentesis, such as have been observed by Trousseau and others. If the migrating clots had been more numerous and the cerebral embolism more extensive, that is, if cerebral arteries of more importance or in greater number had been plugged up by them in the case just related, the patient might have been killed outright by the cerebral embolism. Hitherto, rapid death preceded by a few moments of agony and struggling has, in such cases as the foregoing, been attributed (and the autopsy has sometimes justified the view) to obstruction of the pulmonary artery by an autochthonous clot, (thrombus,) or by an embolus from the right side of the heart; whilst sudden or instantaneous death has been explained by syncope, resulting from reflex irritation of the nerves of the heart. The latter hypothesis is admitted in default of a better one, and in the absence of any appreciable lesion.

M. Vallin remarks that if, in his patient, the apoplectiform attack had proved immediately fatal, he would probably have overlooked the cerebral lesion; and thus this case would have been classed as one of death from syncope. In cases belonging to this category, therefore, it becomes very desirable that, at the autopsy, the brain and its vessels should be examined with sufficient care.

M. Vallin has sought in works of authority for similar cases of pleurisy attended with apoplectiform symptoms followed by cerebral softening, but has not gained much information. He found, however, in the *Bulletin de la Société Anatomique* of 1861, the case of an aged female, who in the course of an acute pleurisy was seized with hemiplegia, and died in twenty-four hours. The autopsy showed an occlusion of the anterior cerebral artery by a fibrinous clot, but without any appreciable lesion of the cerebral substance. The left ventricle of this patient contained old fibrinous clots. In the *Army Medical Reports* for 1859, he found a case similar to his own. This patient was a soldier, who, in the course of a pleurisy with a vast amount of effusion, was suddenly attacked with apoplexy attended by hemiplegia; six weeks afterwards he succumbed to emphysema; at the autopsy circumscribed softening of the brain was found, which had destroyed the whole of the corpus striatum. Unfortunately the arteries of the brain were not examined. (Vide *Gazette Hebdomadaire*, No. 5, 1870; *Half-Yearly Abstract of Med. Sciences*, vol. li., pp. 1–3, 1870.)

CASE XXXVI.

Pleurisy attended with apoplexy and hemiplegia of left side; death three months afterward from exhaustion; autopsy; embolism of basilar artery; right cerebral hemisphere extensively softened; false-membrane, the product of a rather recent inflammation, found in the right pleural cavity; reported by Dr. ABERCROMBIE.

A youth, aged 18, had been affected with cough and pain in the chest for six or eight weeks, and was supposed to be phthisical; but for several days he had been much better, when on Dec. 15th, 1819, he suddenly fell down deprived of sense and motion, and paralytic on the left side, with twisting of the mouth. When partially recovered, he complained of severe pain in the right temple; speech very indistinct; countenance expressive of great stupor. He was not much benefited by treatment. However, in January, 1820, he improved so as to have less uneasiness in his head, and considerable use of his leg, but his arm remained entirely paralyzed. On February 15th, he complained of pain in the back of his head, and suddenly lost his speech and the power of swallowing. He soon recovered his speech, but the power of swallowing was permanently gone, so that from this time he had to be fed with liquids introduced into the stomach through an elastic tube. He died rather suddenly March 20th, having the day before become extremely weak and pale without any obvious cause.

Autopsy.—On removing the dura mater, there appeared on the middle of the right hemisphere a remarkable depression, which, when cut into, was found to be due to an extensive mass of pure ramollissement; the part being in the state of a soft white pulp, without any appearance of pus, and without fetor; it extended the whole depth of the hemisphere. In the cerebral matter adjoining this disease there was a small abscess, no larger than a bean, lined with a firm, soft cyst of coagulable lymph. There was very little effusion into the ventricles, and no other disease in the substance of the brain.

On raising the brain, a remarkable appearance was found in the basilar artery; it was very much enlarged and hard for about an inch in extent, and this portion was found to be completely filled up with a firm white substance having no appearance of blood. Anterior to this portion, there was a small clot of blood in the artery.

The lungs were tolerably healthy; but there was a considerable deposit of coagulable lymph, forming a thick, firm mass betwixt the right lung and pleura costalis at the lower part immediately above the diaphragm. (Vide *Abercrombie on Diseases of the Brain*, pp. 89–91.)

Comments.—That this young man had pleurisy at the beginning of his sickness is proved by the symptoms which he then presented, and by the fact that at the autopsy his lungs were found to be tolerably healthy, while the right pleural cavity contained the products of pleuritic inflammation in abundant quantity and of corresponding date. This case, therefore, belongs to the same category as the one reported, and the two others referred to by M. Vallin, which are mentioned above. In all probability, there was copious effusion of serum in this case also, and its absorption occasioned the decided improvement in his symptoms which was noticed for several days prior to December 15th, when he was attacked with an apoplectic stupor and hemiplegia, due to embolism of the basilar artery. The embolus, doubtless, migrated from either the left side of the heart or the pulmonary veins; but more probably from the latter. It may have consisted of one of those coagulæ which are prone to form in vessels where the blood stagnates, and had its origin in one of the pulmonary veins as a consequence of obstruction of the pulmonary circulation due to compression of the lung from pleuritic effusion. When the lung expanded on the withdrawal of this fluid by absorption, and the blood again passed freely through the capillaries from the terminal branches of the pulmonary artery into the radicles of the pulmonary veins, this coagulum may have become detached and carried away in the current of the circulating blood until finally it chanced to lodge in the basilar artery, where it terminates in the posterior cerebrals, plugged up or occluded the right one of these vessels, and laid the foundation for secondary thrombosis and that extensive obliteration of the basilar artery which was revealed by the autopsy.

The most important feature of this case, in a practical point of view, is the connection which existed between pleurisy or pleuro-pneumonia, on the one hand, and the occurrence of cerebral embolism, on the other; for the former occasioned death, in this instance, only by inducing the latter. We can readily understand that, if the middle cerebral artery had been plugged up by an embolus simultaneously with the posterior cerebral, death might have much more speedily followed the embolism; and that, in any case, if the vertebrals and internal carotids were simultaneously occluded by coagula, death would be instantaneously produced, for in such a case the whole brain would be suddenly rendered anæmic. Cerebral embolism always paralyzes the nerve-fibres and ganglion-cells throughout the part of the brain which it deprives of nutrient blood; therefore, whenever it happens to obstruct a sufficient number of the cerebral arteries at the same time, or to involve the arteries which supply the medulla oblongata with blood, whether others are occluded or not, sudden death must inevitably ensue. Thus, by inducing cerebral embolism, pleurisy may terminate in sudden death. Now, it is a matter of fact that patients having pleurisy with effusion not unfrequently die very suddenly and unexpectedly. Trousseau says: "Sudden death may be one of the consequences. It is not unusual, I repeat, for persons with extensive pleuritic effusion to sink all at once without having had the breathing much oppressed at any time, and without ever having had a threatening of suffocation. Death takes place from syncope. In corroboration of this statement I appeal to the cases, published by my professional brethren, of sudden death occurring under such conditions as I have now described. I could also adduce several similar cases which have occurred in my own practice." . . . . . "Perhaps death is sometimes induced [in these cases] by the formation of clots in the heart and large vessels, which is liable to occur from the circulation of the blood being impeded. This opinion, which I announced long ago, has been verified by a case to which Dr. Blachet has directed attention. (Vide *Union Médicale* for February, 1862.) This physician relates the interesting case of a patient who died suddenly in a faint. At the autopsy a clot was found occupying the trunk of the pulmonary artery in its entire extent, and, bifurcating, it stretched into the divisions of the third and fourth order of the left branch of the artery. In this case the pleurisy was chronic; the heart was not displaced, and the sudden death was probably the result of the blood coagulating in the pulmonary artery." (Vide *Lectures on Clinical Medicine*, vol. iii. p. 221, New Sydenham Soc. ed.) Trousseau also relates a case in point which occurred in his own practice. A male subject, gouty from his youth, had pleurisy on the left side with much effusion. The operation of paracentesis thoracis was indicated and performed with much benefit. There was no symptom which foreboded a fatal termination. But the patient, says Trousseau, was a man of very violent temper. Notwithstanding my formal orders to the contrary, he left his bed to go to stool. He got up, took some steps, then sat down on the convenience, and after some minutes spent in unavailing efforts, he returned to bed. Again he tried, but fruitlessly. He felt great oppression of the breathing, but he declared that he should make one more attempt. Neither the advice nor the entreaties of his family availed to induce him to desist. He resolutely got out of bed, sat on the night-stool, where for some time he made new and unavailing efforts; he then regained the side of his bed, and, when attempting to get in, he expired." (Vide *op. cit.*

vol. iii. p. 280.) Trousseau supposes that death was produced by syncope; but is it not much more probable that it resulted from cerebral embolism, and that the embolia came either from the left side of the heart or from the pulmonary veins, having become detached by his persistent efforts in straining at stool? M. Vallin, whose case we have related above, very justly remarks that if, in his patient, the apoplectiform attack had proved immediately fatal, he would probably have overlooked the cerebral lesion, for a certain time is required for the arrest of circulation to produce any appreciable change in the appearance of the brain; and thus his case also would have been classed as one of death by syncope. It is necessary, therefore, not to conclude too hastily as to the apparent integrity of the cerebral tissue in the absence of any obvious vascular obliteration. It is probable that if, in the cases of sudden death which have occurred during the course of pleurisy, an examination of the brain and its vessels had been made with sufficient care, embolism of the cerebral arteries would have been found in a majority of instances. This subject is of great practical importance, and this must be our apology for according so much space to its discussion.

But *pneumonia* may also give rise to cerebral embolism. When it does, the coagula which plug up the cerebral arteries migrate either from the left chambers of the heart or from the pulmonary veins, where they have been formed in consequence of the inflammatory process in the pulmonary tissue. Double pneumonia would be more likely to induce this preliminary thrombosis in the pulmonary veins, or in the left auricle of the heart, than single pneumonia, because the former embarrasses or retards the flow of blood in these vessels more than the latter. It is not improbable that in the following case of pneumonia, related by Mushet, the apoplectic symptoms were due to cerebral embolism: A patient "with coma and stertor, admitted into the St. Marylebone Infirmary, was seen by a well-known physician, who diagnosed hemorrhage into the brain, and indeed designated the locality. After death the only morbid appearance, on strict examination, was extensive double pneumonia. There was no reason to suspect that this was secondary to typhus, albuminuria, or otherwise intercurrent." (Vide *op. cit.* p. 105.) It does not appear in this case that the arteries of the brain were examined for embolia. Moreover, the apoplectic phenomena and the post-mortem lesions, as far as observed, afford good ground for suspecting that this was really a case of apoplectiform cerebral embolism.

The following case, which Dr. Loomis related to the N. Y. Pathological Society, February 23d, 1870, is an instance in which pneumonia gave rise to, or was the starting-point of, cerebral embolism. This patient died suddenly, and quite unexpectedly, during convalescence from inflammation of the right lung.

Case XXXVII.

Pneumonia ; convalescence ; sudden seizure with hemiplegia, pallor, pulselessness, and gasping breathing ; death. occurred in fifteen minutes ; autopsy ; embolia found in middle cerebral and coronary arteries ; clot in aorta, etc.

A Swedish sailor, aged 28, was admitted to Bellevue Hospital, February 2d, with the following history: Three days previously, he had been seized with a sharp chill, followed by constriction about chest, some difficulty of breathing, some pain in side, with cough, slight expectoration, and fever. These symptoms grew worse up to the time of his admission. Then his temperature was found to be 103½°, his pulse 100, his sputum was such as characterizes pneumonia, and physical examination detected that disease at the apex of the right lung. On the next two days his temperature rose to 104°, and his pulse was a little over 100. On the third day, his temperature fell to 99°, and his pulse was about 80. His sputa changed, his dyspnœa was relieved, and he was fairly convalescent. He was not regarded as seriously ill at any time. His convalescence was rapid and satisfactory, and he was soon up and around the ward. On the fourteenth of February, twelve days after admission, he suddenly died. He was seen by the house-physician about an hour before death, and appeared to be doing well. The urgent symptoms came on fifteen minutes before death, and he was pulseless before the house-physician could reach him. He seemed to be paralyzed on the right side, that is, he had hemiplegia. His face was pale, and his respiration gasping. No signs of heart-disease had at any time been observed.

Autopsy twenty-four hours after death.—A plug (embolus) was found in a branch of one (the left ?) middle cerebral artery. The other portions of the brain were normal. A firm plug was also found in each coronary artery of the heart. One of them seemed to be continuous with a thrombus, having the size of a hickory-nut, which lay just above the aortic valves. The lungs were congested and œdematous; the right one was also firmly bound down with pleuritic adhesions. The liver, spleen, and kidneys were healthy, except one of the latter, embedded in the substance of which were small yellow deposits. Dr. Loomis also stated that the left ventricle of the heart was not distended, containing only a little fluid blood, but no clots; that the patient had not been reduced much by his sickness; and, in reply to a question, that the heart had been examined during life, but without discovering any symptom of cardiac disease. (Vide *The* (N. Y.) *Medical Record*, vol. v. pp. 40, 41.)

Comments.—The autopsy in this case of sudden death during convalescence from pneumonia clearly showed that cerebral embolism had occurred, for a plug was found in one of the cerebral arteries. It is also probable that the cerebral embolism assisted not a little in producing the sudden change for the worse in this man's condition, as it was noticed that he had hemiplegia, and the sudden occurrence of hemiplegia in this case cannot be satisfactorily accounted for by any hypothesis except that of cerebral embolism. The embolia which were found in the coronary arteries of the heart were likewise concerned in producing the fatal result. Indeed, it is not difficult to conceive how they may have lent a powerful assistance in bringing about such a result. This case, then, is an instance in which an apoplectiform attack was, in all probability, produced by simultaneous embolism of the cerebral and coronary arteries, which in turn had its starting-point in pneumonia.

Gangrene of the lungs sometimes induces cerebral embolism. In such cases the embolia (plural of the Latin word *embolium*, which signifies "something thrown in") consist either of shreds of broken-down pulmonary tissue, which have been taken up by the pulmonary veins, and have been launched by them into the systemic circulation, or of coagula which have become detached from the pulmonary veins themselves, or from the left chambers of the heart.

Phthisis pulmonalis occasionally induces cerebral embolism. Colrat has read a paper before the Société des Sciences Médicales at Lyons (*Lyon. Méd.* 1869, ii. 609) on a case of hemiplegia with symptoms of cerebral embolism which occurred in a patient suffering from phthisis. Perroud gave the notes of three other cases of the same kind, and Tripier of another still alive, affected probably in the same way. The autopsies of these are given. Perroud considers that embolism occurring in phthisis originates in one or other of the following ways: 1. Endocarditis. 2. Cavities from which the broken-down lung-tissue in shreds has passed into the circulation. 3. Detached coagula from the left heart. The great tendency to hyperinosis observed in phthisical patients supports the last hypothesis. (Vide *New Sydenham Soc. Retrospect* for 1869–70, p. 112.) It is probable that some at least of the cases of hemiplegia occurring in the course of pulmonary phthisis which have been observed by Andral and others, and have been ascribed by them to cerebral congestion, were in reality cases of cerebral embolism.

Dr. Markham has recorded in his work *On Diseases of the Heart* a remarkable instance in which a woman of 50 was suddenly seized with apoplectic stupor and hemiplegia, and died in sixty hours. The innominate, right common carotid, left internal carotid, and middle cerebral arteries were found to be obstructed with coagula. (Vide *Mushet on Apoplexy*, p. 142.)

In the cases of cerebral embolism which we have thus far considered, some artery or arteries of considerable size, such, for example, as the carotid, basilar, anterior cerebral and middle cerebral, or arteria fissuræ Sylvii, were found to be plugged up with embolia. But there is another important class of cases in which arteries of much smaller size, capillary arteries in fact, become obstructed in this manner, and furnish us with examples of capillary cerebral embolism. The following case is an instance of this sort.

CASE XXXVIII.

Melancholia from occlusion of numerous minute arteries of the brain, with embolia derived from the mitral valve; also commencing hæmatoma of the dura mater.

DR. JOFFE, of the Vienna Lunatic Asylum, has related a remarkable case. A woman, aged 46, was seized with melancholia. She had enjoyed good health until three years before, when she lost her husband and her two children. She had always led a temperate life. She was suffering from delusions, such as that she had no blood and no bowels; that therefore she need not eat; that she would live forever, but that her soul was lost. Organs of respiration healthy. There was a systolic murmur over the left ventricle; the second pulmonary sound was accentuated; the cardiac somewhat increased; mitral insufficiency was evident; appetite poor; bowels constipated, sleep quiet; the lower limbs rather cold, skin sallow, sensibility very much dulled; irritation of skin produced no reflex movements; the sense of smell seemed almost altogether wanting, but she complained of a foul taste. She was mostly apathetic, and sat immovably still, only occasionally suffered for a few days from anxiety, and then cried and complained. Pulse 90, and somewhat fuller at such times, and she ate no food while the excitement lasted. After ten months of treatment, she was strikingly better as regards her mental symptoms. One night soon after this she suddenly awoke from sleep with a cry, and declared that she was everlastingly damned. Large doses of narcotics produced no effect. Great depression soon set in; pulse slow, and hardly to be felt; consciousness much obscured; her strength increased somewhat in the next few days, but she obstinately refused food; the skin was altogether insensitive. Toward the end of the month, her lower extremities became scorbutically colored; a few days later, profuse diarrhœa set in, and soon carried her off.

On *dissection*, the skull-walls were found very thick and dense, the dura mater materially distended, its inner surface lined with false membranes containing spots of blood over the convexity of the hemispheres. The inner membranes were thin; the subarachnoid fluid in large quantity, and the hemispheres much wasted; cortical substance pale, medullary substance moist and soft; a few drachms of clear serum in the ventricles; in the pericardium a few drachms of clear fluid; heart soft and its tissue fatty; the free edge of the mitral valve and the tendinous cords around it were shortened and thickened; in the neighborhood of the free border was a circle of tough, pale-red vegetations; fluid and slightly coagulated blood in the cavities of the heart.

Microscopic examination of the false membranes on the dura mater showed connective tissue-like matter, with new vessels and hemorrhage. The arachnoid, especially on the convex surface of the cerebrum, was spotted with molecular formations; considerable patches were thickened with a fibrous formation, containing many roundish or oval cells, and often with granular nuclei. In many places, there were bodies with concentric layers, containing in the middle pale molecules of various sizes; these bodies arranged themselves in numerous polypoid swellings on the arachnoid, which at their edges frequently showed a division, and in the interior a number of small, pale bodies of various forms.

The vessels of the pia mater were half as large again as usual, contracted at some points, and much contorted, and filled with dark-red blood. The dark-red contents disappeared from the arterial vessels immediately on section, and the rest of the vascular stem down to its branches was filled with yellowish-white molecular masses, obviously of the nature of embolia. Usually an embolus was found at the bifurcation of a vessel, and extended a long way into its branches, without, however, filling these or the trunk of the vessel, so that there was no complete obstruction to circulation. The embolia were sometimes more and sometimes less yellow in color, and of a more or less minute molecular structure, and of varying firmness. These embolisms were found on the convex surface of the frontal and posterior lobes of both cerebral hemispheres, and were of such a kind that a piece of pia mater taken from the foremost part of the frontal lobe showed under the microscope embolia in only a few vessels, but in the neighborhood of the central anterior convolutions there were embolia in numerous vessels. In the tissue of the arachnoid and between the vessels there was a quantity of brownish-red pigment, and also in the ventricles, where the vessels were bordered with a broad hyaline margin, and reddish pigment. The vessels of the pia mater were dilated even where there was no embolism. Many capillaries of the pia mater were thickened and surrounded with fine fibres. In one place, the capillary artery and all its twigs were enveloped in connective tissue. At some places the cortical substance showed its arteries dilated and filled with embolic masses. The embolism of the brain-vessels was of the same kind as that found in the vessels of the pia mater. The nerve-cells of the cortex in the vicinity of an embolism were partly atrophied, partly filled with fatty bodies; and deposits of finely granular fat were found around embolic deposits. The finely granular nucleated substance of the cortex was cloudy, dark-colored, and in many places filtrated, resembling the appearances found in general paralysis. The spinal cord was also much altered; the central gray matter showed, at the lumbar swelling and in the lower part of the dorsal region, only a striking pigmentation of the large cells of the anterior horns. From the lower two thirds up to the cervical enlargement the gray substance was morbidly changed. Both at the gray commissure and in the horns, the gray matter surrounding the central canal was overlaid with numerous amyloid bodies, which were densest throughout the whole gray commissure.

It is plain that embolism, produced by dislodgment of matter from the mitral valve, was the cause of all the brain symptoms. The patient had had two attacks of melancholia before, from which she had improved, but never entirely recovered. (Vide *New Sydenham Soc. Retrospect*, 1867–8, pp. 74–76.)

Etiology of Cerebral Embolism.—In the cases which we have related this affection was produced by simple endocarditis and the organic lesions consequent thereon, and by pleurisy or pleuro-pneumonia, but mainly by the former. It has also been known to be induced by pneumonia, by pulmonary gangrene, and by pulmonary consumption. Furthermore, we should not overlook the influence which ulcerating endocarditis, and ulcerating endarteritis, not unfrequently exert in the production of this disease. The disintegration of tissue which attends these affections may result in the detachment of shreds and granules which are washed away by the circulating blood and ultimately become embolia. The spontaneous bursting of atheromatous, fibrinous, or purulent cysts of arteries into their calibre, and the discharge of their contents into the circulating blood, may also furnish embolia. But in cases of cerebral embolism the plugs with which the arteries are closed generally consist of detached concretions of fibrin, that have been deposited on the roughened parts of the valves in consequence of endocarditis or valvular disease, or else of portions of the valves themselves, they being in a state of ulcerous inflammation, which have been washed off by the current of the blood. Sometimes, however, these plugs have their origin in necrotic foci in the lungs, and in thrombosis of the pulmonary veins or left chambers of the heart, as we have already shown. In one very instructive case, published by Esmarch, an embolus obstructing the internal carotid came from an aneurism of the common carotid, having been set free by the manipulations during an examination. (*Niemeyer.*)

Embolia vary in size from those which suffice to plug up the internal carotid or basilar, down to those which can occlude only the capillary branches. The latter are called capillary embolia; and generally result from the breaking up of the clots, and the disintegration of the fibrin that has been deposited on the cardiac valves. Sometimes they result from the bursting of the atheromatous and other cysts into the calibre of the arteries, as mentioned above. Besides, there are some cases of capillary embolia in which the starting-point is ulcerating endocarditis, as Rokitansky, Virchow, Bamberger, and Freidreich, in Germany, as well as Charcot and Vulpian, in France, have shown. (Vide *Trousseau's Lectures on Clinical Medicine*, vol. iii. p. 419.) Proust, however, does not believe that granular matter, the contents of cysts, or of abscesses in the heart, which have spontaneously ruptured, can produce an embolism of the capillary arteries of the brain. He speaks of one class of capillary embolisms, the existence of which is undoubted, and in which the obstructing matter exerts a morbid influence over and above its mechanical effect; for example, the embolisms which are formed from the matter of a gangrenous lung. Such an embolism, he thinks, is apt to induce local gangrene. Weber, however, rejects the idea that such embolia produce any thing more than purely mechanical effects. (Vide *New Sydenham Soc. Retrospect*, 1865–6, p. 103; also "*Des differentes Formes de Ramollissement du Cerveau.*" *Thèse de Concours*, par *Dr. Adrien Proust.* Paris, 1866.) In addition to the above-mentioned varieties of capillary embolia, the so-called *pigment* embolia, according to Meissner, deserve special mention. They are met with in bad cases of malarial fever, even in our climate. These pigment embolia are not unfrequently formed with great rapidity, so that after some violent paroxysms of fever, with delirium, convulsions, and even maniacal phenomena, death takes place with stertor and coma, from miliary embolism of an apoplectic character. Sometimes the cerebral symptoms do not occur until the fever has lasted a long time. Sometimes the whole process is chronic, with or without notable functional disorder. According to the results of autopsies, the pigment in most cases is formed in the spleen, whence it is carried to the liver, where the larger flakes are detained, while the smaller ones penetrate through the pulmonary capillaries, and pass in the arterial stream to all parts of the body. In the brain, however, such large pigment-flakes are often found that they could not have passed through the hepatic and pulmonary capillaries, but must have originated in the blood itself. The deposit of pigment in the cutaneous capillaries imparts that earthy tint to the skin which is so often observed in chronic cases of malarial disease. Pigmentary embolism occurs less frequently in the brain than in the spinal cord, where it may occasion either temporary paraplegia, or, if attended with extensive infarctions and atrophy, permanent contractions and palsies. (Vide *New Sydenham Soc. Year-Book*, 1861, p. 187; also *H. Meissner in Schmidt's Jahrb.*, vol. cix., p. 89.) But Meissner holds that in most cases the cause of capillary embolia is cardiac disease of some kind or other, and especially that form which he designates endocarditis ulcerosa acuta. It runs its course with typhoid or pyæmic symptoms, and is especially apt to occur in debilitated subjects having acute rheumatism, and in feeble women in the puerperal state. The ulcerations in these cases are almost always seated in the valves of the left ventricle, occasionally in those of the right; they result from an inflammatory molecular decay of the endocardium, and induce, by the roughness of their surface, the formation of fibrinous deposits, which are subsequently washed away by the circulating blood, and plug up the arteries in remote parts. (Vide *New Sydenham Soc. Year-Book*, 1863, pp. 140, 141.)

Cerebral embolism may occur at any period of life. It is, however, met with much more frequently before the middle epoch than after it. For example, the age is given in seventeen of the cases which we have related or referred to in this chapter. Of them one was 13, one 18, one 21, two 24, one 28, one 29, one 30, one 32, one 33, two 34, one 46, one 47, one 51, one 52, and one 78. Twelve were less than 35, and only five above that age. Besides, one patient was described as a young married lady, and another as quite a young man, making in all fourteen out of nineteen, or almost three fourths, who were less than that age. This remarkable predilection of cerebral embolism for the early periods of life is probably to be accounted for by the fact that rheumatic endocarditis occurs more frequently in the young than in the aged.

Anatomical Appearances produced by Cerebral Embolism.—The morbid appearances presented by the brain in cases of this disorder vary according to the size and number of the occluded arteries, and the length of time that the patient has survived the attack. The first effect of plugging a cerebral artery, whether it be a main trunk or only its capillary branches, with embolia, is always to deprive the brain-substance to a corresponding extent of its nutrient blood. But the appearance of the brain-substance itself is not the same in the two cases. When an artery like the middle cerebral is plugged up at its origin, or the anterior cerebral just above the anterior communicating, or the posterior cerebral just after giving off the posterior communicating, the portion of brain-substance most affected is generally found to present a pale, anæmic, or bloodless appearance, surrounded, however, by a congested border. But when the embolism is capillary in character, that is, when the capillary arteries only are occluded with embolia, the portion of brain-substance affected is usually found to present a congested or hyperæmic appearance. MM. Prevost and Cotard think they have established, by experiments on animals with artificially produced embolism, that a manifest congestion is always produced in the parts to which the obliterated artery is distributed. (Vide *New Sydenham Soc. Retrospect*, 1865–6, p. 100.) But autopsies in recent cases of cerebral embolism occurring in the human subject have also shown that when a comparatively large artery, such, for example, as the middle cerebral, is occluded at its origin, more or less pallor or anæmia is produced in the corresponding portion of the cerebral substance. (See Dr. Kirkes's first case, already referred to, and others.) With regard to the effect of capillary embolism on the cerebral substance, Proust notices the remarkable fact, above mentioned, that the immediate consequence of this occurrence is to produce a hyperæmia or passive congestion of the part whose blood-supply is interfered with. This circumstance has also been noticed by Cohn, Vulpian, Rokitanski, and Moreau. Proust thinks that a complete explanation of this fact is at present impossible; but it is very striking, and has an interesting confirmation in the circumstance that, in cases of red softening, the foci are habitually in relation with an embolism or a thrombosis. Occasionally, however, a patch of ramollissement is found to be pale, from the first, and this generally depends on the complete plugging up of a large arterial trunk, as stated above. (Vide *New Sydenham Soc. Retrospect*, 1865–6, p. 100–102; also *Etudes physiologiques et pathologiques sur le Ramollissement Cérébral*, par *J. L. Prevost* et *J. Cotard*, etc., Paris, 1866; and *Des differentes formes de Ramollissement du Cerveau*, etc., par *Dr. Adrien Proust*, Paris, 1866.)[1] We think that the hyperæmic appearance of the brain-substance, which is met with in cases of capillary embolism, can be accounted for, at least in part, by the vaso-motor paralysis that soon occurs in the coats of the occluded vessel and all its branches, from want of nutrient blood, and by the flow of blood in some considerable quantity from the adjacent sound capillaries into the dilated or paralyzed ones, where it speedily stagnates. Or, in other words, we think this hyperæmic appearance results from an attempt on the part of nature to establish a vicarious circulation in the affected portion of the brain, but which proves to be unsuccessful, because the walls of the obstructed vessels are paralyzed, their calibre dilated, and the blood speedily becomes stagnant in them. The affected part looks congested because its capillaries are all passively dilated and filled with stagnant blood. This hypothesis seems to satisfactorily account for the hyperæmic appearance which occurs throughout the portion of brain-substance affected in cases of capillary embolism, and in the periphery of the part affected in cases where a large trunk is plugged up with embolia. Thus we find that the first morbid appearance which the brain presents in cases of cerebral embolism is either a state of anæmia in the centre of the affected part, with hyperæmia in its periphery, or a state of hyperæmia alone, according as the occluded vessel consists of a large arterial trunk, or of a number of arterial capillaries. It is, however, probable that the brain generally presents an exsanguinated or bloodless appearance in those cases where death occurs instantaneously, or after the lapse of only a few minutes, in consequence of cerebral embolism. MM. Prevost and Cotard have conclusively shown that the cerebral hyperæmia mentioned above, as induced by capillary obstructions, etc., is not inflammatory in its nature. As we have already stated, it is purely passive in its origin and character.[1]

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[1] A specimen of white softening of the left cerebral hemisphere was exhibited to the New-York Pathological Society, December 14th, 1870. It was obtained from a female patient having hemiplegia, who died at Charity Hospital. This change was thought to be due to cerebral embolism, as some branches of the middle cerebral artery were found closed from atheroma. The examination, however, could not be made with a view to determine the exact situation of the plug. The heart was in a nearly normal state, but there was a slight deposit of atheroma in the ascending aorta. (Vide *The* (N. Y.) *Medical Record*, vol. v., pp. 547, 548.)

Again, if death does not occur till the lapse of some days or weeks after the attack, the portion of brain to which the occluded artery or arteries ordinarily furnish the blood-supply is always found to be undergoing a process of softening or necrosis. This form of cerebral softening is analogous to the gangrene of the extremities which is induced by obstruction of their main arteries. In both cases the death of the affected tissues is due to arrest or deprivation of the supply of nutritive material; but the necrosed parts within the skull, not being exposed to the action of the atmosphere, are but seldom decomposed. This happens only when the embolia which plug the vessels migrate from a suppurating or gangrenous spot, and bring with them a tendency to suppuration or gangrene. Embolism of a cerebral artery induces necrosis of the brain-substance more readily the later and more incompletely a collateral circulation is established. But whether or not the closure of a cerebral artery by an embolus shall induce necrotic softening depends chiefly on the seat of the obstruction. If, as rarely happens, there be anæmia in a part of the brain chiefly supplied by an artery which is occluded by an embolus before reaching the circle of Willis, for example, the internal carotid, it will generally pass off soon and necrosis will not be produced; but if an important artery be closed by an embolus above the circle of Willis, necrosis is the usual termination; so great is the influence which the circle of Willis exerts in equalizing the cerebral circulation.

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[1] When a somewhat large fibrinous clot passes into one of the carotid or vertebral arteries, it causes the formation of hemorrhagic foci of minute size in the brain, that is, cerebral infarctions, or else gives rise to extreme anæmia of the brain-substance, and consequent necrosis of the anæmic portion, according as the artery that becomes plugged by the clot (embolus) has a small or a large size. The symptoms always are suddenly developed. If the part of the brain thus deprived of blood has a considerable size, the symptoms are those of an apoplectic stroke, and, in bad cases, the patient may be quickly or even instantaneously destroyed. (*Niemeyer.*)

It is remarkable that, in cases of cerebral embolism, the artery obstructed much more frequently than any other is the middle cerebral of the left side. Perhaps this is partly due to the fact that the left carotid artery leaves the arch of the aorta almost in the direction of the blood-current, while the innominate forms a considerable angle with it. (*Rühle.*) The seat of softening is usually found in the cerebral hemispheres, and in the left much oftener than in the right. It varies in size from that of a bean to a goose's egg, and sometimes is still larger.

The softening which occurs in the brain as the result of embolism, or deprivation of the affected part of nutrient blood, may be *red*, *yellow*, or *white*, according to the stage in which it is examined. *Red* softening commences after twenty-four or forty-eight hours, dating from the apoplectiform attack, and lasts from eight to fourteen days. It is characterized by a moderately diminished consistence, a red, brownish, or rosy color, sometimes united with a slight tumefaction of the tissue, and always with increased tension and injection of the collateral vessels. Under the microsocope, the nerve-fibres and ganglion-cells appear partly normal, partly broken down and granular; the capillary walls for the most part unchanged; sometimes, however, they are filled with coagulated blood. *Yellow* softening does not appear till after the lapse of fourteen days. The necrosed patch of brain-substance is by this time more pappy; the nerve-fibres and ganglion-cells are broken up, dislocated, granular, scarcely distinguishable, and in a state of retrograde fatty metamorphosis. The capillary walls are covered with gray fatty granules; they contain wasted blood-corpuscles, and especially granular hæmatin from the shrunken red globules, and also enlarged granular white corpuscles. The yellow color depends on the latter, and on the fading of the red globules from loss of their hæmatin, and sometimes also from the formation of fat. *White* softening generally does not appear until several months have elapsed. (Occasionally, however, it is present from the very outset, as we have already stated.) The affected part of the brain is then completely white, diffluent like cream, with some whitish flocculi suspended in it. The nerve-fibres and ganglion-cells have all disappeared, the capillaries and blood-corpuscles have become metamorphosed, and under the microscope we see numerous granules, oil-drops, and granular cells, which give the liquid a very strong resemblance to the colostrum. This third phase is met with only in large patches of softening, for the small ones are always absorbed. Sometimes, even in large patches, the softened mass becomes absorbed, which constitutes then a fourth phase. It is characterized by depression of the cerebral surface, and by the formation of cysts and cicatrices. It also tends to result in recovery.

An important peculiarity of cerebral softening, when produced by embolism, is, that it never affects the whole region belonging to the obstructed artery, but only its central part, on account of the influence which is exerted by the collateral circulation.

Cerebral softening, when induced by plugging up of the cerebral capillaries, differs from that which has just been described, by the existence of small scattered foci on the surface or in the interior of the cerebral substance; in the softening which results from obstruction of the venous sinuses, that is, from thrombosis of the sinuses of the dura mater, there are the same small scattered foci, corresponding in their distribution to the course of the affected sinus, and mingled with numerous capillary hemorrhages or extravasations, besides which the softened pulp usually has a red color.

Cerebral softening may also be produced by inflammation of the brain. It is characterized by the formation of genuine pus, as well as other inflammatory products, with induration of the surrounding brain-tissue, and sometimes by the development of arachnoid adhesions or false membranes.

Again, cerebral softening may be induced by the occurrence of plastic exudation in, or by hyperplasia of, the cerebral connective tissue, as a result of constitutional syphilis, that is by the formation of gummy deposits in the brain. (Vide *Lancereaux's Treatise on Syphilis*, vol. ii. pp. 52–70, New Sydenham Soc. ed.)

Lastly, cerebral softening may be occasioned by cerebral tumors, or by extravasations of blood, in a kind of mechanical way. All these latter forms of softening are, however, of comparatively infrequent occurrence, as Lancereaux in twenty-two cases found sixteen to depend on obstruction of the cerebral vessels in the nature of either embolism or thrombosis. (Vide *H. Meissner's Report on Thrombosis and Embolism in New Sydenham Soc. Year-Book* for 1863, pp. 139, 140; also *Schmidt's Jahrb.*, vol. cxvii., p. 209.)

In cases of embolism, the walls of the affected artery are, for the most part, unaffected as far as the unaided eye can perceive; but Lebert has, at the end of nineteen days from the attack, found with the microscope a considerable increase of the connective-tissue corpuscles, and a division and multiplication of the nuclei. Grisolle also found thickening of the arterial coats and adhesion of the same to the embolus in a case where a calcareous fragment driven into the middle cerebral artery had excited inflammation and fibrinous deposits there. The embolus is variously shaped, but mostly fusiform or rounded; it is generally colorless or whitish, sometimes, however, gray or brownish; and it is almost always dry, hard, and resisting. It is mostly fibrinous, or is separable into two parts; one peripheral, which is always fibrinous, and sometimes very long; and the other central, directed toward the heart, which is calcareous, atheromatous, or fibrinous also, and sometimes contains heterogeneous elements, such as warty growths or bits of the cardiac valves. Sometimes the embolus cannot be found, having probably become absorbed; and in cases of long standing the obstructed vessel itself occasionally disappears. (Vide *H. Meissner*, *op. cit.*)

One of the most striking changes, however, which the embolus is found to undergo is "canalization." The following is an instance in point: *Dr. Wilson Fox*, at the Pathological Society of London, introduced an example of embolism of the middle cerebral artery in which "canalization" had been accomplished, one part of the artery, however, being still occluded. The man temporarily recovered through the partial reëstablishment of the circulation. *Dr. Murchison*, who had seen the patient, stated that he was suddenly seized with pain in the right side of the head, followed quickly by paralysis of the left side of the body. The man had had acute rheumatism. (Vide *Lancet*, 1867, vol. ii. p. 487.) In another case admitted to St. George's Hospital, under *Dr. Ogle's* care, October 2d, 1867, the left internal carotid artery, just below its bifurcation, was found blocked up by a fibrinous plug that adhered firmly to the walls of that vessel, and was perforated or tunneled by an opening or canal which readily allowed the passage of a bristle. (Vide *Lancet*, February 29th, 1868, p. 287.) In some cases the canalization appears to be due to the fact that by contracting or shrinking the plug withdraws itself from one side of the artery; or, if it happens to be strongly adherent to the arterial walls, it may give way in the middle, so as to form a central perforation. In other cases, however, the canalization results from the spontaneous softening or liquefaction of the fibrin in the interior of the plug. In this way the flow of the blood in its normal channels is allowed in part to be resumed. Canalization, when present, constitutes the first step in the absorption of embolia and thrombi.

But embolism of a cerebral artery is not unfrequently attended with thrombosis of the same artery. For example, an embolus lodges in the middle cerebral at a point where some important branches are given off, but does not plug it up completely, although the current of blood through it is greatly retarded, whereupon the stagnant stream of blood itself coagulates around the embolus until the occlusion becomes complete, and sometimes this process of clotting continues till the obstructed vessel becomes entirely filled with coagula from its origin on the one hand to its several terminal branches on the other. This is precisely what happened in the fourth of Trousseau's cases which we have already briefly sketched. In that case the embolus was small, but it formed the starting-point of a thrombosis which soon filled up the middle cerebral artery, and all its branches with clotted blood. In Dr. Abercrombie's case also, (vide *supra*, Case XXXVI.,) embolism of the basilar artery was, in all probability, attended with the formation of a good-sized thrombus, which became organized, and when the autopsy was held, two and a half months subsequent to the attack, the walls of the artery were found to be thickened, and its calibre filled up, to the extent of an inch, with a firm white substance. Why does arterial thrombosis occur in some, but not in all of the cases of cerebral embolism? The answer is, that the blood, in consequence of hyperinosis or of some unknown change, has acquired a morbid tendency to coagulate in some but not in all of the cases belonging to this category. In Trousseau's case, for example, this abnormal coagulability of the blood was due to the puerperal state.

But the cerebral arteries sometimes become occluded with coagulated blood in consequence of thrombosis alone, or at least without the presence of any embolia. As a rule, these thromboses form at points where, as a result of chronic endarteritis, or, as is usually said, of atheromatous degeneration, the calibre of the cerebral vessels is diminished, and the inner wall roughened. It is only exceptionally that the blood spontaneously coagulates in cerebral arteries whose walls are healthy, that is, that marasmic arterial thrombosis occurs. (*Niemeyer.*) Thrombosis of the cerebral arteries is an affection which belongs especially to the advanced periods of life, because atheromatous degeneration belongs especially to those periods of life, while embolism occurs most frequently during the early periods.

Thrombosis of one or more of the arteries of the brain may, however, occur in consequence of acute arteritis or endarteritis of a local character, as it did in the next three cases which we shall relate. This variety of arterial thrombosis of the brain is met with quite as frequently during the early and middle as during the advanced periods of life.

CASE XXXIX.

Hemiplegia of right side with temporary loss of consciousness suddenly occurred without apparent cause; death forty-eight days afterward with symptoms of cerebral softening; autopsy; middle cerebral artery on each side inflamed and occluded by a thrombus; left corpus striatum softened and discolored, etc.

Joseph T——, a seaman, born in England, aged 29, admitted to the Brooklyn City Hospital, N. Y., (services of Drs. H. S. Smith and James

Crane,) October 2d, 1865. History meagre. A friend said the patient had been feeling poorly for a few days, and was lying down, when he suddenly lost power of the whole right side, and suffered a temporary unconsciousness. No traumatic cause. No exposure to heat or effects of alcohol. Never had rheumatism. Had no premonitory head-symptoms. On admission, had facial paralysis in addition to palsy of the right arm and leg; face drawn to left side; tongue turned to right when protruded; partial loss of power in right orbicularis palpebrarum; partial loss of ability to articulate; voice very thick; sensation and temperature good on right side as well as on left; inclined to somnolency; evacuations natural.

Until Nov. 13th he slowly improved. He protruded his tongue more directly, could move his leg and foot a little, and had some slight use of his arm. His bowels were generally very sluggish, but when freely moved, he was much brighter. The inclination to sleep continued.

Nov. 13.—A change occurred. The *left* side of his body now became cold, while the right side retained its natural warmth. No loss of motion on left side.

Nov. 14.—Very dull; did not respond when addressed; left conjunctiva inflamed; bowels inactive.

Nov. 15 to 17.—Comatose; respiration stertorous and labored; face flushed; head hot; body motionless.

Nov. 18.—Died without change of symptoms.

Autopsy.—Both middle cerebral arteries were found occluded by firmly adherent clots at points close to their origin from the internal carotids. Arteritis had existed in each vessel, which led to thickening of its walls and the formation of a thrombus. The clot on the left side was larger than the other, and quite pale. The clot on the right side was dark, and extended a short distance into the anterior cerebral artery. By the microscope no atheroma was found in the arterial walls. The left corpus striatum was softened, and darker in color than natural. Condition of other organs not stated. The case was reported by *Dr. Francis H. Atkins*, House-Physician. (Vide *The* (N. Y.) *Medical Record*, March, 1866, p. 19.)

Comments by the author.—This was probably a case of thrombosis or spontaneous coagulation of blood in the middle cerebral arteries, and it probably resulted from inflammation of these vessels, with thickening of their walls and corresponding diminution of their bore. The thrombus on the left side was considerably older than the other; for it was larger in size and quite pale in color. The red corpuscles had had time to disappear to great extent. The thrombus on the right side was still dark in color and recent in appearance. The thrombosis of the left middle cerebral artery in all probability occurred at the commencement of his attack, and gave rise to the apoplectic phenomena, the hemiplegia of the right side, and the softening of the left corpus striatum. The thrombosis of the right middle cerebral artery, on the other hand, began on the 13th of November, that is, some forty-three days subsequent to the attack, and soon produced the comatose condition which terminated in death five days afterward. This case possesses very great interest. The arteritis which induced the blood to coagulate in the middle cerebral arteries appears to have occurred without any ascertainable cause. At least it was not due to injury, nor to exposure to heat, nor to the abuse of alcohol, nor to the rheumatic diathesis; and the seizure with hemiplegia, etc., was not preceded by any symptoms of a warning character.

Cases XL. and XLI.

Sudden death in both with apoplectic symptoms; thrombosis of the basilar artery resulting from inflammation of the walls of that artery; the cause, etc.

Two interesting cases of rapid death from occlusion of the basilar artery are reported by M. Hayem. They both present striking analogies. The patients were both females; one was thirty-three years old, and the other fifty-two. The histories were incomplete; both women, however, died very suddenly with symptoms of intense apoplexy, and nothing was found to account for death but thrombosis of the basilar artery.

The walls of this vessel were found to be the seat of inflammatory changes that were quite characteristic, and may be described as follows:—Commencing at a point in the walls of the artery there was developed a thickening (swelling) having considerable depth at its centre and a diffused margin. This swelling involved all three tunics, but the inner one most of all. Over this swelling the adventitious coat was found to be abnormally vascular; in one of the cases mentioned above large vessels could be seen with the naked eye. The exterior of the artery was embossed, and its calibre diminished; it looked white, opaque, horny; its consistence was firm like that of fibro-cartilage. On making a transverse section, the swollen side of the artery was seen to be five or six times thicker than the opposite side, and, on pursuing the examination, the excised slice was found to consist of several distinct layers separated apparently by exudation. These swollen laminæ pushed the lining membrane of the artery inwards, and then, after bursting it, projected into the blood passing by the rent, and induced it to coagulate. If this swelling be more minutely investigated, the preëxisting elements in all parts of it may be seen enlarged by a granular exudation, and possessing abnormally two, three, or even a greater number of nuclei, and externally to these elements a profusion of new elementary forms. Thus, there is interstitial exudation and multiplication of the elements of the vascular walls. Though this process is an inflammation of the walls of the artery, it is not simply an endo-arteritis or peri-arteritis; the pathological changes extend through all three tunics, presenting, among others, characteristic alterations of the middle coat. In fact, the middle coat is the seat of an abundant multiplication of new elements, and the smooth fibres themselves take part in this new formation. Thus, from an anatomical point of view, a form of arteritis is presented in these instances which is remarkable from its being seated in the three coats and from its inflammatory nature being undoubted. (Vide *Half-Yearly Abstract of the Medical Sciences*, 1868, vol. xlvii. pp. 190, 191; also *M. G. Hayem in Archives de Physiologie Normale et Pathologique*, No. 2, 1868.)

In the above-mentioned cases of thrombosis of the basilar artery reported by M. Hayem, the inflammation of the walls of that vessel caused the blood to coagulate in it:—1. Because the swelling of the tunics which resulted from their inflammation diminished the calibre or contracted the bore of the artery at the place of disease to a corresponding extent; 2. Because after the internal coat had been ruptured by the swelling, a rough or jagged surface was presented to the blood-stream on the interior of the artery at the same place, instead of the smooth surface that is natural; and 3. Because the blood came into immediate or direct contact with the inflamed structures which constitute its walls.

The history of the last three cases shows conclusively that the phenomena of apoplexy, in the correct sense of the term, may be induced in young or middle-aged subjects by thrombosis or the spontaneous coagulation of blood in the arteries of the brain. The history of these cases furthermore shows that when thrombosis of the cerebral arteries occurs in young or middle-aged subjects it is generally due to an acute arteritis of a circumscribed character, but involving all the arterial tunics at the same time, instead of resulting from chronic endarteritis, or so-called atheromatous degeneration of the inner tunic, as it usually does in the aged.[1]

Finally, we have to state that the plugging of a cerebral artery in consequence of embolism or thrombosis, or both combined, occasionally gives rise to cerebral or intra-cranial aneurism. In the *Medical Record* for March, 1869, pp. 1, 2, Dr. Echeverria has reported a case, illustrated with a plate, which belongs to this category. The subject was a man, aged 67, an epileptic from his youth, who died suddenly while straining at stool. The autopsy showed that hemorrhage from a ruptured aneurism of the right vertebral artery was the cause of death. But the left vertebral was also affected with an aneurism of similar size, that was still unbroken. Thus, there were two aneurisms involving the terminal portions of the vertebral arteries. Further examination showed that each of these vessels were completely plugged up by a clot at the point where it unites with its fellow to form the basilar, and that this occlusion had given rise to the double aneurism. These arteries, together with those at the base of the cerebrum, were in a state of fatty and atheromatous degeneration. Their walls were therefore proportionally weakened and less able to withstand the effects of intra-vascular pressure. The cerebral substance appeared dry and bloodless. The pons Varolii was covered with a thin film of coagulated blood, extending into the spinal canal; the medulla oblongata and spinal cord were embedded in a mass of grumous blood, which had escaped from the ruptured aneurism.

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[1] Thrombosis of the cerebral arteries is only incidentally mentioned above as a substantive disease, and cases belonging to the apoplectiform variety of it alone are presented. The author, however, expects soon to publish an essay on cerebral thrombosis in which the other varieties of it will receive suitable mention.

Heart enlarged, atheromatous deposits in the valves; arch of aorta extensively changed, with large calcareous and atheromatous patches. During life his pulse was 80, irregular and small; there were signs of cardiac hypertrophy, with rough aortic direct murmur; urine normal. Some symptoms referrible to the aneurism had existed for several months before his death. They were a feeling of dizziness on attempting to walk, impaired ability to move or protrude the tongue, difficulty in swallowing, and sometimes he would lose the power of speaking aloud, and had to whisper words with considerable effort. The roots of the glosso-pharyngeal and hypoglossal nerves were compressed by the aneurismal swelling of the right vertebral artery. This artery also separated into two branches for a short distance, at the seat of aneurism, and through the hole thus formed the spinal accessory nerve passed. As the aneurisms increased in size, the symptoms mentioned above grew worse. His mind, however, was clear up to the time of death.

It is probable that in this man's case one or more clots migrated from the left side of his diseased heart to the posterior vertebral arteries, and induced a secondary thrombosis which occluded them at their place of junction, as well as the basilar artery, and thus laid the foundation for aneurismal expansion of their walls already stiffened and weakened by fatty degeneration. Thus, in occasional instances, cerebral embolism produces cerebral aneurism, and ultimately death from cerebral or intracranial hemorrhage.

Symptoms and Course of Cerebral Embolism.—Partial or circumscribed anæmia and necrosis of the brain, when due to embolism, are almost always preceded by characteristic premonitory symptoms. But these are not brain-symptoms; they are those of the diseases which almost exclusively cause embolism of the cerebral and other systemic arteries, that is, of valvular disease of the heart, of endocarditis, of pleurisy, of pneumonia, or of some other destructive disease of the lungs. The occurrence of these premonitory symptoms, and the presence or absence of valvular disease, endocarditis, pleurisy, or severe disease of the lungs, have such an effect upon the diagnosis between embolism of a cerebral artery and other brain-diseases, that with the same set of symptoms we may diagnose embolism with tolerable certainty, if we find them,[1] and exclude it with great certainty if they are absent. The sudden shutting off of arterial blood from the part of the brain supplied by the obstructed artery, instantly arrests its functional power. Experience shows that embolia generally lodge in the middle cerebral artery, (arteria fossæ Sylvii,) and particularly the left one; as the sudden closure of this large artery causes great anæmia in the parts of the brain supplied by it, we can readily see that sudden hemiplegia, especially of the right side, is the most important symptom from which to diagnose cerebral embolism, if it occur in a patient having valvular disease of the heart, etc. The entire loss of consciousness and the apoplectic phenomena which usually attend the hemiplegia at the outset, when the middle cerebral artery is plugged up by an embolus, is hard to explain. But it is evident that hemiplegia, occurring suddenly and attended with apoplectic stupor, the result of cerebral embolism, might readily be mistaken for the effects of cerebral hemorrhage. In some cases, the age of the patient affords some grounds for distinguishing a hemorrhage from an embolism. Hemorrhages occur chiefly, although not exclusively, in advanced age, embolism is found in persons of any age; hence, in young persons, the presumption is in favor of embolism. However, the principal way of avoiding error is afforded by the careful examination of the heart, lungs, and other organs. The probability which the diagnosis receives from the discovery of valvular disease of the heart, etc., is greatly strengthened if we can find a coincident embolism of a peripheral artery or of one of the internal organs, such as the spleen or kidney.

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[1] Mistakes have, however, been made. For example, in the case of Mary Murphy, which we related in the last chapter in order to illustrate Broca's views concerning the dependence of *aphasia* upon some lesion of the third frontal convolution of the left hemisphere, it was found at the autopsy that her hemiplegia and other cerebral symptoms were due to cerebral hemorrhage, although she had valvular disease of the heart of an old date, and her paralysis and aphasia were attributed to cerebral embolism, until the autopsy was held, without the existence of hemorrhage in the brain being suspected. Hammond also mentions a case of mitral regurgitation, under his charge, in which cerebral embolism was diagnosticated, but extravasation into the corpus striatum was discovered to be the cause of death. Besides, Hughlings Jackson (*Brit. Med. Jour.* Oct. 1870) has reported a case in which there was cerebral hemorrhage with hemiplegia, together with extensive valvular disease of the heart. (Vide *Hammond on Diseases of the Nervous System*, pp. 132, 133.)

In most cases, death occurs sooner or later after the attack, with the symptoms of general paralysis. In a good many cases, however, the consciousness returns after a time; but the symptoms of paralysis (hemiplegia) generally do not disappear. This is sufficiently explained, as we have already shown, by the obstacles which, in most cases, exist to the establishment of a collateral circulation. (Vide *Niemeyer's Text-Book of Practical Medicine*, vol. ii. pp. 183, 184, 1st Am. ed.)

The history of the following case will serve a good purpose for illustrating the symptoms which are present, and the principles upon which the diagnosis is founded, in many instances of cerebral embolism.

Case XLII.

Chronic valvular disease of heart; hemiplegia of left side and apoplectic coma suddenly occurred; partial recovery; death about three months afterward from cardiac and renal dropsy; no autopsy.

Mrs. S——, aged 51, married, and in good circumstances of life, had suffered from valvular disease of the heart for more than twelve years. The first sound of that organ was accompanied by a harsh bellows murmur, which was loudest near its apex. She had difficulty of breathing, which was increased by muscular effort, as in going up-stairs, etc. With the lapse of time the general symptoms of mitral regurgitation gradually became more strongly marked. In the fall of 1865, her dyspnœa became very troublesome, and she began to have anasarca in the feet. At the end of November she caught a heavy cold while riding on a bleak road and on a cold, windy day. It was attended with some inflammation of the pulmonary tissue, as well as bronchitis, and left behind it a harassing cough, with a strong tendency to pulmonary œdema. After this attack she never went down-stairs again.

In January, 1866, about the middle of the month, while sitting in her easy chair conversing pleasantly with her family, one evening at about 8 o'clock, she was suddenly seized with apoplectic stupor and hemiplegia of the left side. Without any warning she suddenly sank back in her chair in a state of unconsciousness, and began to slide down toward the floor, and would have fallen thereon if her husband had not caught hold of her and prevented it. At the same time it was noticed that her mouth was drawn toward the right ear, and that her breathing was choking and stertorous. On attempting to give her some brandy, it flowed out of the left or paralyzed corner of her mouth. Her countenance was very pale and the surface of her body cold. Her pulse was small, weak, and frequent. She lay utterly unconscious till midnight, when reaction having come on, she began to show some signs of coming to herself, but did not seem able to recognize any body until the next day. The left side of her body proved to be completely paralyzed as to motion, and partially so as to sensation; that is, she had left-side hemiplegia. She had also entirely lost the faculty of speech. At the end of three days, however, she could articulate enough to be understood with great difficulty; but afterward she improved so much that she could talk pretty well, although her tongue continued to be rather thick at times. After the attack her mind remained permanently affected. Before it she was remarkably clear-headed. Thus she suddenly became childish and silly. Her appetite became ravenous and uncontrolled by judgment. But her facial palsy disappeared in about a month, so much that it was scarcely observable, unless she attempted to laugh. The paralysis of her extremities also slowly improved, and in about two months she got so much better that she could walk into the next room with her daughter's assistance, but dragging her paralyzed foot after her. On being questioned about it, she said that when the attack occurred she felt exactly as if somebody had struck her on the forehead with a billet of wood and knocked her senseless, and she even imagined, and firmly believed, too, that one of her acquaintances, who was absent, had done it. She died, April 6th, 1866, of dropsy from cardiac and renal disease, and preserved her consciousness to the last. No autopsy.

Comments.—This was not a case of cerebral hemorrhage. The apoplectic stupor came on much too quickly for that, as we have already shown. (See *Chapter on Cerebral Hemorrhage.*) If the attack had been produced by extravasation of blood in the brain, she would, in all probability, have perished on the spot. There is no good reason for doubting that this was a case of cerebral embolism. The symptoms were exactly those which this disorder often occasions. She had long exhibited premonitory phenomena in the shape of disturbances induced by the valvular lesion of her heart. Moreover, she had suffered a premonitory embolism of the left brachial artery, while traveling, about eighteen months before the final attack. Then she suddenly lost the use of her left fore-arm and hand. It was attended with great pain, although the sensibility of these members was likewise lost. This paralysis, however, disappeared entirely in two or three days, on the establishment of the collateral circulation. It is probable that in the last attack the middle cerebral artery on the right side was more or less completely plugged up by an embolus, which had migrated from the left side of her heart. She came very near to perishing at the outset of this attack. If the embolism, or the cerebral anæmia resulting from it, had been but little more extensive or profound, she would have been killed on the spot. Cerebral embolism not unfrequently occasions sudden death. It is probably the proximate cause of the fatal issue in most of those cases where valvular disease of the heart produces instantaneous death. It is probable that if, in such cases, the cerebral arteries were examined with sufficient care, some of them would generally be found plugged up with embolia.

Death was produced very suddenly in the two cases next to be related. They gave one or more shrieks, and immediately, or almost immediately, expired. In the author's opinion cerebral embolism was the immediate cause of death.

(*Alcoholic intemperance, ascites, anasarca, and albuminuria ; patient convalescent when he died very suddenly without apparent cause ; autopsy ; the fibrinous products of recent endocarditis found on the mitral valve.*)

William Dalton, aged 35, born in Ireland, said to have been much addicted to drinking, was an inmate of the Emigrant's State Hospital on Ward's Island. When admitted to hospital he had hydrops abdominalis or ascites, anasarca of the lower extremities and scrotum, together with albumen in the urine. His system readily responded to treatment, the dropsical accumulations were almost gone, and he seemed to be convalescing satisfactorily in every respect, when he suddenly died on the morning of October 21st. He gave a shriek while lying in bed, and, without presenting any other symptom of disease, immediately expired.

Autopsy, by the author, thirty-six hours after death.—Left lung strongly adherent throughout to parietes of chest. Upper lobe of same lung and superior part of lower lobe thickly sown with miliary tubercles. Right pleural cavity contains about four ounces of serum. Right lung nearly healthy. The abdominal cavity contains about three quarts of reddish-colored serum. Liver exhibits fatty degeneration in an early stage. Spleen softened. Stomach much increased in size or capacity ; its mucous membrane is softened in every part. (The patient had a voracious appetite, delighting especially in eating large quantities of soup and bread.) Kidneys softened ; middle third of each much disorganized. Heart enlarged and very flabby ; mitral and tricuspid valves exhibit the marks of recent and severe inflammation ; aortic valves healthy ; pericardium contained about four ounces of liquid, but there were no signs of pericarditis. The head unfortunately was not opened, for the importance of this proceeding in such cases was not at that time generally recognized. It is, however, not improbable that if the cerebral arteries had been examined, they would have been found extensively blocked up with fibrinous coagula which had been washed off from the mitral valve of the heart.

(*Sudden death, occasioned probably by cerebral embolism; autopsy; mitral valve encrusted with old fibrinous vegetations; extensive extravasations of blood found in the substance of the liver and pancreas; these organs are also very much softened, etc.*)

William Brophy, aged 64, married, and of good habits, died very suddenly at his home in Twentieth street, near the Sixteenth Ward Station-House, on the night of May 26th. He was by trade a house-carpenter, had worked all the previous day as usual, and went to bed without making any complaints. His wife says he was awake at two o'clock, but did not complain of any thing. She also declares that a little while afterward he uttered some piercing shrieks, exhibited slight convulsive movements, and almost immediately expired. She had gone to sleep in the mean time, and was suddenly awakened by his screams. For several weeks before death, his wife noticed that his appetite was failing, that he could take scarcely any breakfast, and that he was getting thin. Still he continued to work at his trade every day as if he were in perfect health. He was an Irish emigrant, and had lived in New-York about fifteen months.

Autopsy, by the author, thirteen hours after death.—Cadaver in a good state of preservation, emaciated, pale, and slightly jaundiced, (yellow;) pupils natural; rigor mortis strong. *Thorax.*—About lower lobe of right lung some old pleuritic adhesions were found; left lung entirely free from them; quantity of blood in lungs normal, but it has gravitated into their posterior parts, and is still fluid; tissue of lungs normal in every respect save a little excess of pigmentum nigrum. Heart normal in size and shape, somewhat fatty on its exterior, and flabby; mitral valve exhibits an abundance of old white fibrinous vegetations on the free margin of each segment; tricuspid valve exhibits similar vegetations on its edge, but they are not nearly so abundant; valves of pulmonary artery normal; aortic valves thinner than natural toward their free margin; endocardium, except the vegetations mentioned above, normal in color and in every other respect; pericardium also natural; lining membrane of the arteries perfectly natural; foramen ovale completely closed. The blood was free from clot or entirely fluid, and natural in color in the heart and in all the vessels examined, as it generally is in cases of sudden death. *Abdomen.*—Liver normal in size and shape, but its color is darker than natural; on its convex surface are numerous spots which are almost black, and look like those seen on the skin in cases of purpura hemorrhagica; on further examination they are found to be produced by blood (still fluid) effused beneath the peritoneal coat and capsule of the organ; this covering or investment is itself normal in every respect, but it is peeled off from the organ with very great ease; more easily, indeed, than in any other case that has come under my observation, and on making but slight traction; the substance of the liver is uniformly very much softened in every part thereof; it breaks down under slight pressure; it then becomes a pulp-like, grumous mass, that is, quite moist with bloody fluid; the hepatic blood-vessels, especially the large ones, are not softened; the color of the hepatic tissue is darker than natural; the softening is so well marked that a medical gentleman witnessing the autopsy suggests that the hepatic substance might be washed away with a stream of water so as to leave scarcely any thing besides the blood-vessels, ducts, nerves, and fibrous tissue behind; gall-bladder rather more than half-full of bile, which is normal in consistence, but darkened in color; its mucous membrane is thickened, not softened, but rather firmer than natural in consistence. Pancreas reddish-black in color, on a large part of its exterior, from blood effused beneath its fascia or capsule. Its substance is also very much softened, and on slicing it the cut surface is found to present a dark marbled appearance in every part, from the presence of extravasated blood, which has not penetrated the different interstices alike. No plastic exudation whatever found in or about the pancreas and liver. No smell of putrefaction exhaled. The spleen is enlarged and softened. Its peritoneal coat is thickly speckled with firm, old, grayish-white, granular, fibrinous deposits. The surface of a section has a dirty bluish-brown or almost black color. The small whitish strings or trabeculæ which are always found in the substance of the healthy spleen in great numbers, are not visible in this case. The kidneys are normal in size. Their capsules exhibit opaque spots of variable size, which are dry and resemble parchment. The kidneys are indurated and non-elastic in feel, and brittle in consistence. On stripping off the capsule from the left one, its surface is found to have a decidedly granular appearance. The cut surface of either organ presents a reddish-brown color, and is quite dry. The cortical substance is thinner than natural. There is a deposit of firm, old, whitish, fibrinous exudation at the base of one pyramid in the left kidney. The right organ shows fewer signs of disease than the left. The bladder contains a little urine. The stomach holds some food partially digested which exhales the odor of onions. Mucous membrane of stomach and intestines reddened in spots, located principally on the folds, in consequence of venous hyperæmia. The head was not opened, and I now regret this omission exceedingly, for it is probable that if the cerebral arteries had been examined with sufficient care, they would have been found to be extensively obstructed with embolia derived from the mitral valve of the heart.

Comments.—The morbid appearances revealed by the autopsy in this case have been related with considerable minuteness, because of their exceptional or unusual character. Atrophic softening of the liver and pancreas is not often met with, and spontaneous extravasation of blood into these organs is equally rare. Can this softening and hemórrhage be satisfactorily accounted for, on the hypothesis that they were produced by embolism of these organs? I think not, because the softening was of a uniform grade in every part of the liver, on the one hand, and of the pancreas, on the other, and this could scarcely have obtained if the softening had resulted from interference with the blood-supply by embolia. Under such circumstances, some parts of these organs would, in all probability, have been found in a more advanced stage of softening than others. Moreover, if the disorder of the pancreas was really due to embolism, how happens it that the spleen's condition was not the same as that of the pancreas, for both of these organs are mainly supplied with blood by the same artery?

The opinion that death in this case was produced by cerebral embolism is, in the absence of anatomical proof, founded on the fact that embolism of the cerebral arteries, if sufficiently extensive, would occasion sudden death, that embolia might readily have been derived from the mitral valve of the heart, and that the subject exhibited one striking symptom, which is not unfrequently observed in cases of cerebral embolism, namely, he uttered loud cries. This symptom was noted in the following case of cerebral embolism:—

Dr. Fagge showed to the Pathological Society of London, October 18th, 1870, a specimen of embolism of the middle cerebral artery, of six weeks' duration, taken from a woman of twenty-six, admitted to Guy's Hospital, September 5th. On the day of admission *she gave a cry*, and suddenly became insensible, being paralyzed on the right side. Next day she became conscious, but could never be got to say more than "Yes" or "No," subsequently. She sank into a drowsy condition, and died violently delirious. A loud systolic murmur was heard over the heart, and the liver was enlarged. The brain *post-mortem* showed its left apex atrophied and softened, and an embolus was found in the middle cerebral artery. There were also embolia in the kidneys and spleen, and numerous vegetations on the valves of the heart. (Vide *Lancet*, 1870, vol. ii. p. 607.)

It is not difficult to understand why the victims of cerebral embolism utter one or more loud cries when attacked. When embolism occurs in the extremities it is generally attended with severe pain. There is no good reason why embolism of the brain also should not produce intense pain. It was noted in one of Trousseau's cases of this disease, mentioned above, that "she suddenly felt very acute pain in the right side of the head." It is probable that this symptom would be noted more frequently if inquiries concerning it were specially made. When very acute pain in the head suddenly occurs, it is very natural for many persons to give loud cries. Children almost always do so.

With regard to the symptoms of cerebral embolism, Meissner states that when it occurs in arteries on the proximal side of the circle of Willis, the circulation is, for the most part, soon restored by means of the free anastomoses belonging to that circle; but when the distal ones are affected by it, the results are usually permanent. When the carotid is plugged with embolia, the symptoms are sudden loss of consciousness and hemiplegia, often accompanied by general convulsions. These symptoms soon disappear as the supply of blood to the brain is restored by the collateral circulation. When, on the other hand, mere peripheral arteries, such as the anterior, middle, or posterior cerebral, are affected, the consequences are much more serious. Such patients mostly drop down unconscious, as if struck with apoplexy, and death may immediately ensue. If they recover consciousness, hemiplegia, blindness, deafness, or loss of speech remains, or most violent eclampsic and maniacal attacks set in, succeeded at a later period by monotonous delirium. Complete recovery but seldom or never takes place, even after the establishment of a collateral circulation; generally, the phenomena of encephalitis supervene, which may prove quickly fatal. Recovery, as far as existing statistics show, is possible only when the collateral circulation is established before forty-eight hours have elapsed from the occurrence of the obstruction. Recovery may be complete when one carotid trunk is obstructed; but it is never perfect when the obstruction is situated on the further side of the circle of Willis. A partial recovery, a cyst, or a cicatrix having been formed in consequence of absorption of the softened part, is often attended with a peculiarly unintelligible articulation. Pigmentary embolism, since it affects the capillaries of the gray substance almost exclusively, impairs the mental functions much more than the locomotive. If the pigmentary flakes accumulate rapidly, death mostly ensues with typhoid phenomena, or those of icterus gravis, and the gray substance is found covered over with numerous, minute extravasations of blood, (so-called apoplexia disseminata,) each of which contains in its middle a flake of pigment. In chronic cases there is a rather gradual impairment of the intellectual functions, or the plugging of the capillaries remains quite latent, until an accidental attack of congestion leads to the extravasation of blood. (Vide *New Sydenham Soc. Year-Book* for 1861, p. 188.)

Diagnosis of Cerebral Embolism.—There is a strong resemblance between the symptoms produced by embolism of the cerebral arteries, by thrombosis of the same arteries, and by extravasation of blood into the substance of the brain; and therefore it is often correspondingly difficult to make a diagnosis between these affections. Cerebral hemorrhage and arterial thrombosis both occur most frequently in old persons. Moreover, the disease of the arterial walls, the chronic endarteritis, which leads them to be ruptured in the one class of cases, is the same as that which induces thrombosis in the other class. Between hemorrhage into the substance of the brain and thrombosis of the arteries of the brain the diagnosis is generally very hard to make. Bamberger says that, in his notes, he finds seven cases where there was an error of diagnosis, and the real state of affairs was only discovered at the autopsy; he considers it impossible to avoid this error, and says he seldom ventures to make an absolute diagnosis of cerebral hemorrhage. (*Niemeyer.*)

There is, however, a much better chance to diagnose cerebral embolism, on the one hand, from cerebral hemorrhage and arterial thrombosis of the brain, on the other; and attention to the following points will afford material assistance: 1. The symptoms of hemiplegia and apoplectic stupor are almost always suddenly developed at the commencement of the attack in cases of cerebral embolism; while in cases of cerebral hemorrhage or cerebral thrombosis, these symptoms are generally developed more or less slowly, that is, one after another, and not all at once. (See *Chapter on Cerebral Hemorrhage.*) 2. Cerebral embolism is always preceded by characteristic premonitory symptoms, as has been pointed out by Niemeyer and others. These symptoms pertain to the several diseases which almost exclusively occasion embolism of the cerebral arteries, namely, valvular disease of the heart, endocarditis, pleurisy, pulmonary inflammation, pulmonary consumption, and pulmonary gangrene. Cerebral hemorrhage and cerebral thrombosis are, for the most part, not preceded nor attended by the phenomena of these diseases. When hemiplegia, with or without apoplectic stupor, occurs suddenly in the course of either of the diseases above-mentioned, it is almost certain that some artery of the brain has become plugged up with embolia. 3. The age of the patient may be an important item for consideration; rheumatic endocarditis and ulcerating or destructive endocarditis are diseases of early life, while cerebral hemorrhage and arterial thrombosis belong mostly to the later periods. 4. The sudden occurrence of acute pain in the head at the moment of attack, denoted by screams or otherwise, may also be an important item for consideration. Romberg states that cerebral hemorrhage is much less frequently accompanied by headache than any other cerebral disorder. Embolism of the cerebral arteries, on the other hand, often produces, all of a sudden, very acute pain in the affected part of the head, as we have shown above.

Again, the ophthalmoscope can, not unfrequently, be employed with advantage, in making a differential diagnosis of cerebral embolism. On this point Dr. Hammond justly says: "In examining a case of recent embolism, the ophthalmoscope should always be used to view the fundus of the eye, and even in old cases valuable signs will often be obtained. The middle cerebral artery, the ordinary seat of embolus, arises from the internal carotid after the anterior cerebral and ophthalmic have been given off. Occlusion of its channel must, of course, throw an increased amount of blood into the last-named arteries, and, as the arteria centralis retinæ is derived from the ophthalmic, it and its branches become enlarged. The ophthalmoscope will enable us to discover the congestion thus produced, and will often be the means of helping us to determine, in the absence of paralysis, which side of the brain is the seat of the lesion. In older cases we will frequently find retinal congestion." (Vide *Hammond's Treatise on Diseases of the Nervous System*, pp. 129, 130.)

The following case, believed to be an instance of cerebral embolism, is presented to the reader because the symptoms in it were rather unique or different from the general run, and because the ophthalmoscope was advantageously employed in its elucidation. Moreover, it appears to be the first case on record in which the ophthalmoscope was employed for that purpose.

(*Acute rheumatism followed by valvular disease of the heart; headache, strabismus and deviation of mouth and tongue, which were attributed to cerebral embolism, afterward occurred; recovery; the ophthalmoscope was used with advantage in diagnosticating and watching the progress of this case.*)

John Turnbull, aged 17, admitted to the Hull General Infirmary April 25th, 1867. He was tall, much wasted, and had a suffering expression, with converging strabismus of left eye, and the mouth very slightly drawn to left side; pulse 70, and very thrilling; a large coarse systolic murmur was heard near left nipple. He was perfectly sensible, and complained of severe frontal headache, with confusion of vision. He stated that his condition had been much the same for seven weeks, that his illness began spontaneously with headache and vomiting, without loss of consciousness or convulsions, and that in the previous summer he had had an attack of acute rheumatism. He was ordered grs. iij. of blue mass, and grs. ij. of extract of henbane in a pill, and a draught of acetate of ammonia, three times a day, and spirit-lotion to the head. "No marked alteration in his condition, except progressive debility, took place till May 2d, when he complained of increased headache and dimness of vision, and being unable to expectorate, from excessive weakness, death from bronchial obstruction threatened. With the aid of some champagne, he rallied in about twenty-four hours, and at the end of a week was much improved, having a clean tongue and a good appetite, but the headache, strabismus, and deviation of the tongue to the left remained. On May 16th, it was noticed that these symptoms had passed off, with the exception of the last-mentioned. He was ordered a mineral-acid mixture.

"A week later, as he still complained of some dimness of sight, he was examined with the ophthalmoscope. The retinal vessels were found much enlarged, and the veins very tortuous; the optic-nerve entrance of an intense red color, not being distinguishable from the surrounding parts except by the entrance of the vessels, the redness being chiefly due to a number of very fine vessels radiating from the centre. There was no morbid effusion in any part. He could spell easily from No. 15 of Jaeger's test-types, (being unable to read and write.) He was again examined at the end of another week, when the optic-nerve entrance was observed to be paler in color, so that its circumference could be distinguished, but still much injected, and the vessels nearly as large and tortuous as before; sight was apparently perfect. He was discharged convalescent."

The reporter remarks: "The peculiar form of paralysis in this case denoted some morbid condition within the cranium, which appeared to have its most easy and natural explanation in cerebral embolism, an opinion further supported by the perfect recovery of the patient. The case received much additional interest from the information afforded by the ophthalmoscope, for one may fairly believe that the intense congestion of the retina denoted a similar condition of the brain, perhaps a state of reaction after the circulation had been reëstablished through the collateral channels." (Vide *Hammond's Treatise on Diseases of the Nervous System*, pp. 130, 131; also, *British Medical Journal*, 1867, and *Quarterly Journal of Psychological Medicine*, January, 1868, p. 178.)

Finally, the diagnosis of pigmentary embolism of the cerebral capillaries, in cases where the cerebral functions are suddenly abolished and coma is present, is determined by the fact that the patient has been affected for a more or less considerable period with a pernicious malarial fever. (*H. Meissner.*)

Treatment of Cerebral Embolism.—In cases of anæmia and necrosis or softening of the brain, produced by this cause, there is but small prospect of doing good by treatment. When thrombosis or embolism of the cerebral arteries has occurred, we cannot remove the obstructing coagula with therapeutic agents. Hence the indication would seem to be to favor the development of a collateral circulation, without exposing the patient to new danger from the occurrence of active or inflammatory congestion. It is very difficult to fulfil these indications, and we may readily do harm instead of good. The purer the symptoms of partial paralysis or hemiplegia, the more obstinate they remain; if they are not accompanied by symptoms of cerebral irritation, a tonic and supporting plan of treatment is indicated. Hence we generally find the administration of stimulants recommended in the treatment of softening of the brain. If, on the other hand, symptoms of cerebral irritation caused by active congestion, such as severe headache, muscular contractions, etc., are prominent, it is advisable to apply cold to the head, and perhaps apply leeches behind the ears. We should, however, be very careful about employing venesection in these cases, for it is readily followed by collapse. The treatment must be adapted to the peculiar features of each case. (Vide *Niemeyer's Text-Book of Practical Medicine*, vol. ii. p. 189, 1st Am. ed.) But tonics and nourishing food will be required much oftener than sedatives and a spare diet. In patients who are predisposed to the occurrence of thrombosis and embolism from having pleurisy, pneumonia, or pulmonary consumption we can perhaps do something to prevent it by administering certain preparations of ammonia; for example, if they require a febrifuge, give the acetate or citrate of ammonia; if, on the other hand, they need a stimulant, prescribe the carbonate or the aromatic spirit of ammonia. It is believed that these remedies can exert some influence in diminishing the abnormal coagulability of the blood upon which the occurrence of thrombosis and embolism in these cases largely depends.

CHAPTER VII.

1. ON NERVOUS APOPLEXY: 2. ON CEREBRAL GOUT, ESPECIALLY APOPLECTIFORM CEREBRAL GOUT, OR GOUTY APOPLEXY: 3. ON CEREBRAL RHEUMATISM, ESPECIALLY APOPLECTIFORM CEREBRAL RHEUMATISM, OR RHEUMATIC APOPLEXY.

1. *Definition of Nervous Apoplexy.*—The possibility of its occurrence shown.—Concerning reflex hemiplegia.—*Case* XLIII. Hemiplegia of left side induced probably by intestinal irritation through reflex nervous action; complete recovery in four days on using aperients, etc.—How reflex nervous irritation may suspend the cerebral functions. The phenomena of surgical "shock" explicable on this hypothesis.—*Case* XLIV. Graze-wound of left side inflicted by a cannon-ball; immediate death; the only cause of death discerned was "shock."—"Shock" is not synonymous with nervous apoplexy, but its pathogeny is analogous to that of nervous apoplexy.—Reflex nervous apoplexy. —Was Case XVII. an instance of it? Nervous apoplexy from emotional causes; its symptoms described.—Dr. Copland relates a case of nervous apoplexy which occurred in his own practice.—Names of many other observers given who have reported similar cases.—Proximate cause of nervous apoplexy.—The phenomena of epilepsy, except the convulsions, resemble those of nervous apoplexy. 2. *On Cerebral Gout, and especially Apoplectiform Cerebral Gout or Gouty Apoplexy.*—According to Heberden, the gout disposes its victims to apoplexy.—Drs. Garrod, Tanner, Aitken, etc., state that cerebral gout may take the form of apoplexy.—Dr. Copland relates a case of apoplectic gout —which occurred in his own practice.—Niemeyer on cerebral gout and gouty apoplexy. —Trousseau relates a case of gouty apoplexy.—He relates another interesting case of cerebral gout.—Gouty epilepsy or epileptiform cerebral gout; a case mentioned by Van Swieten; another case related by Dr. Rounel.—Periodical headache or megrim not unfrequently a form of cerebral gout.—Vertigo sometimes caused by gout; a case mentioned.—Occasionally the special senses, for example, that of sight, are impaired in cerebral gout.—Lowness of spirits, vapors, melancholy, hypochondriasis, and other delusions of a similar character, often due to larvaceous or concealed gout.—Certain disturbances of the cerebral functions usually precede attacks of gout; they are described.—Gout probably occasions insanity, epilepsy, apoplexy, and some other forms of cerebral disease much oftener than is generally supposed.—*Nature of Cerebral Gout;* it is generally a neurosis and not an inflammation of the brain or its membranes.—Cerebral hyperæmia generally present in gouty apoplexy; it is liable to become complicated with cerebral œdema; some gouty swellings of the skin described. *Treatment of Cerebral Gout;* lowering remedies, and especially the abstraction of blood to be avoided; when retrocession from some joint has occurred, apply rubefacients to the joint; in bad cases the judicious administration of stimulants often beneficial; general rules for the treatment of anomalous internal gout cannot be laid down; the symptomatic indications in each case should be followed; application of cold to the head, and indications for its employment; potass. bromid., ext. cannabis indica, guaiacum, colchicum, quinia, cerii oxalate, hyoscyamus, opium or morphia, and lithia carb.; the sulphur water of Richfield re-

commended for cerebral gout.—3. *On Rheumatic Apoplexy and the other forms of Cerebral Rheumatism.*—The subject of cerebral rheumatism has recently attracted much attention.—*Case* XLV. Acute multi-articular rheumatism; sudden death with apoplectiform phenomena; no autopsy, but the head symptoms were not due to embolism nor to intra-cranial inflammation.—Trousseau's case of cerebral rheumatism with sudden death.—Cases reported by Drs. Weber, Murchison, Burdon-Sanderson, and Wilson Fox, in which the temperature rose to a great height before death.—The foregoing cases were apoplectiform in character; the autopsies showed that they were not due to cerebral nor pulmonary embolism, nor cardiac thrombosis, nor to inflammation of the brain or its membranes, but in all probability to the action of the rheumatic poison itself upon the brain.—The predisposing causes of cerebral rheumatism stated.—2. *On the Paralytic Variety of Cerebral Rheumatism.*—Several instances of it mentioned by Trousseau.—3. *On Mental Alienation or Insanity as a Variety of Cerebral Rheumatism.*—Griesinger's remarks concerning it.—Sander has reported five cases of mental disease resulting from rheumatism.—Trousseau has related two cases in which chronic delirium was due to cerebral rheumatism.—4. *On the Spasms or Convulsions which are sometimes produced by Cerebral Rheumatism.*—These convulsions may be epileptiform or eclampsic, and tetanic in character.—Cases reported by Drs. Bright, Todd, and Fuller.—5. *On the Choreic Variety of Cerebral Rheumatism.*—What Dr. Bright states, Dr. Sée asserts, and Dr. Trousseau declares concerning it.—A case from Trousseau and another from Thore related.—What Henoch found and H. Roger says concerning the relation between chorea, rheumatism, and cardiac disease.—Dr. Ferber's remarks.—Rheumatic chorea not attended by any structural lesion of the brain or spinal cord.—6. *On the Meningitic Variety of Cerebral Rheumatism.*—It consists of rheumatic inflammation of the cerebral meninges, and is of rare occurrence.—The occurrence of head-symptoms in the course of rheumatic fever must not always make us think that menigitis has occurred.—A number of cases in point referred to.—How rheumatic meningitis may be recognized during life.—All the varieties of cerebral rheumatism are neuroses except the last.—The occurrence of cerebral rheumatism generally not due to metastasis; and proofs given.—*Treatment of Cerebral Rheumatism:* use of derivatives; the symptomatic indications must be followed; n some cases it should be cooling and sedative, in others analeptic and stimulant; wrapping the body in wet sheets; sulphate of quinine and veratrum viride as antipyretics; venesection should not be employed; utility of opium or morphia, potass. bromid., ammon. bromid., potass. iodid., diffusible stimulants and lemon-juice; conium, Calabar bean, arsenic, and anæsthetics for rheumatic chorea; treatment of rheumatic apoplexy.

Nervous apoplexy, gouty apoplexy or apoplectiform cerebral gout, and rheumatic apoplexy or apoplectiform cerebral rheumatism are grouped together and treated of in the same chapter because they are *neuroses*, and in that respect are naturally related to each other.

1. *Definition of Nervous Apoplexy.*—We apply this term to a disorder of the brain in which the cerebral functions are suddenly suspended, not from congestion, nor from œdema, nor from hemorrhage, nor from embolism, but by the operation of certain nervous influences or agencies, which are, for the most part, either emotional or reflex in character. This term has also been applied to those cases of apoplexy in which, on examination after death, no lesion is found to account for the symptoms. Furthermore, the term simple apoplexy has sometimes been erroneously applied to this class of cases.

It has been doubted by some whether there is, in reality, such a disorder as nervous apoplexy. The possibility of its occurrence, however, is proved by the fact that there is such a disease as reflex hemiplegia. For, if an impression in the form of an irritation can be transmitted from some distant part, such, for example, as the colon, through the agency of the afferent nerves, to the brain, and suddenly suspend the functions of one cerebral hemisphere, so as to produce the symptoms of hemiplegia, surely it is possible that a still more powerful impression, when transmitted to the brain by the same means, may suddenly suspend the functions of both cerebral hemispheres, and thus produce the symptoms or phenomena of apoplexy. But does reflex hemiplegia ever really occur? The answer is, that cases of one-sided palsy have not unfrequently been met with whose occurrence and incidental phenomena could not be satisfactorily explained on any other hypothesis than that the palsy was induced through the agency of reflex nervous action. Tanner says hemiplegia "may be due to some distant irritation, such as a colon loaded with unhealthy feces." (Vide *Practice of Medicine*, p. 412, Am. ed. 1870.) Other writers entertain the same opinion; and it is believed that the following case belongs to the same category.

Case XLIII.

Hemiplegia of left side produced probably by reflex nervous action; complete recovery in four days on using aperients, etc.; reported by T. Palmer, M.D.

Mr. A. B——, aged 63, free from all constitutional and organic disease, toothless but in the habit of eating meat, was suddenly seized with left-sided paralysis or hemiplegia in April, 1859. This paralysis improved materially after taking a four-grain dose of calomel; and, after applying six leeches, and taking a few more doses of aperient medicine, disappeared in four days. For four years he continued well, except that he annually had an attack of bronchitis. He also had much anxiety in business. Early in 1863 he began to suffer from nervous disorders, unbearable restlessness and oppression in the early morning, severe nervous dyspepsia, inability to attend to business; in fact, from various symptoms of cerebral irritation. These symptoms were markedly relieved for a time by the use of narcotics, but in a few days their effect was lost, and he was worse than before. But a powerful cholagogue dose of calomel and sulphate of magnesia at last brought away a large quantity of very offensive feces, and the patient was permanently relieved. Dr. Palmer remarks that the result in the second attack shows that the hemiplegia in the first attack was, in all probability, also reflex in its nature. (Vide *New Sydenham Soc. Year-Book* for 1863, p. 107; also *Lancet*, Dec. 19, 1863.)

Comments.—When reflex irritation produces hemiplegia, or any other form of cerebral disturbance, it does so, doubtless, by the influence which it exerts upon the vaso-motor nerves of the cerebral blood-vessels. A strong impression, when transferred from some distant part by reflex action to these vaso-motor nerves, may readily occasion tonic spasm in the walls of the cerebral arteries, with corresponding diminution of the blood-supply, or an anæmic state of the nerve-fibres and ganglion-cells in the affected part of the brain, and corresponding disturbance or even arrest of their functions. Or, in another class of cases, a profound impression, when transmitted in this way, may occasion paralysis of the walls of the cerebral blood-vessels, with corresponding dilatation of their calibre, and a corresponding state of hyperæmia or passive congestion, with stagnation of blood in the disordered portion of the brain, in consequence of which the nerve-fibres and ganglion-cells would fail to receive an adequate supply of fresh arterial blood, and would exhibit corresponding disturbance or arrest of function in the same part of the brain. But, so far as the cerebral symptoms are concerned, the effect of reflex or transferred irritation would be substantially the same, whether vaso-motor spasm of a tonic character or vaso-motor paralysis of the cerebral blood-vessels were induced by it; for in both instances the nerve-fibres and ganglion-cells would fail to receive enough freshly oxygenated blood to maintain their functional activity. There does not seem to be any good reason for doubting that, when reflex irritation produces hemiplegia, it does so by inducing what is really an anæmic state of the histological elements of the cerebral hemisphere belonging to the opposite side of the body. Now, the gist of our argument is that, if reflex irritation can disturb the circulation of blood enough in one cerebral hemisphere to suspend its functions and thus occasion the symptoms of hemiplegia, it is possible that a more profound impression, when reflected to the vaso-motor nerves of the brain, may disturb the circulation of blood in both cerebral hemispheres enough to arrest their functions, and thus produce the symptoms of apoplexy. Furthermore, we not only hold that it is possible for reflex nervous irritation to suspend the cerebral functions, but we believe that, oftentimes, it does do so more or less completely. The cerebral symptoms which attend the so-called "shock" of injury or of surgical operations are, in most cases, explicable on the hypothesis that the local impression is transmitted to the vaso-motor nerves of the brain by reflex action. Indeed, all the phenomena which are produced by the so-called "shock" of injury, etc., are readily explicable on the hypothesis that the local irritation is transmitted by reflex action, not only to the vaso-motor nerves of the brain, but to the whole sympathetic system also. The pallor and coldness of the surface of the body, which are always present when the "shock" is severe, are mainly due to tonic spasm of the cutaneous and other blood-vessels, and corresponding diminution of their contents; the impaired action of the heart to anæmia of the cardiac ganglia, the result of spasm of their nutrient arteries; the loss of consciousness and sensibility to anæmia of the cerebral hemispheres, produced by tonic contraction of the cerebral blood-vessels, etc.; and all these vascular spasms or contractions are to be ascribed to irritation of the vaso-motor nerves in these several parts of the body, transmitted by reflex action from the place of injury. In some instances of "shock" the cerebral symptoms are developed with such suddenness as to bear no inconsiderable resemblance to apoplectic stupor. In rare instances belonging to the same category, the cerebral anæmia is so extensive that all the functions of the brain, including those of the medulla oblongata, are at once suspended, and death immediately ensues. The following case, which occurred in the late war of the rebellion, is believed to be an example in point.

CASE XLIV.

Graze-wound of side, inflicted by a cannon-ball; immediate death; the apparent cause was "shock."

Major Mills, a young officer on the staff of Major-General Humphreys, then commanding the second corps, Army of the Potomac, was killed at the Boydton plank-road, March 31st, 1865, by a cannon-ball. He was on horseback at the time. The projectile grazed the left side of his abdomen, and, passing onward, went through the head of a horse belonging to another officer. The cannon-ball grazed the left side of his abdomen in such a way as to slightly open the cavity and let out some coils of intestine. General Humphreys said the wounded officer rolled up his eyes and immediately fell from his horse quite dead. The author is indebted to Surgeon Charles Page, U. S. Army, at that time medical director of the second army corps, for an account of the case. Dr. Page thinks that death must have been occasioned by "shock," for the wound did not penetrate nor directly involve any organ the lesion of which would cause instant death, *per se*, whatever the secondary consequences might have been if the patient had survived long enough to allow their development.

Comments.—The phenomena which constitute what is known in surgical language as "shock" are, for the most part, not due to the emotion of fear, for they occur in the horse as well as in his rider, when both happen to be severely wounded, and in persons who are unconscious from the administration of anæsthetics, while undergoing surgical operations of a severe character.

The phenomena of "shock" have usually been attributed to weakening of the heart's action, or to diminution in the force with which the blood is propelled through the arteries to the brain. This explanation is insufficient: 1. Because any reflex nervous impression which powerfully affects the heart's action would affect the other parts of the vascular system at the same time, since their functions are presided over by the same ganglionic system of nerves. 2. Because, in many instances, the pallor of the countenance and the disturbance of the cerebral functions are developed more rapidly, and are greater in proportion or degree than the enfeeblement of the heart's action, much greater than we find in cases other than "shock" where the contractions of the heart are equally weak.

It is but seldom that the symptoms of "shock" are developed so suddenly as they were in the case which we have just related. Generally, they do not come on all at once, unless the brain itself is injured, but gradually and more or less slowly, according to the nature of the injury.

We do not claim that the so-called "shock" is ever synonymous with nervous apoplexy, for the range of functional disturbance is much more extensive and involves many more organs in the former than in the latter. What we do claim is, that the cerebral phenomena which are present in bad cases of "shock" are analogous to those which occur in cases of nervous apoplexy, and that in both instances the proximate cause of these cerebral phenomena is the same, namely, anæmia of the nerve-fibres and ganglion-cells of the brain, resulting from disturbance of the cerebral vaso-motor nerves.

We do not doubt that apoplectiform phenomena are sometimes produced by reflex nervous irritation, and that there is such a disease as reflex nervous apoplexy. Case XVII., related in the Chapter on Congestive and Serous Apoplexy, may possibly have been an instance of it. If the apoplectic symptoms in that case were not produced by reflex nervous irritation, they were probably due to cerebral embolism, although no embolia were found, and there were no vegetations nor clots in the heart. The hemiplegia, coma, and death, which occurred in that case, might have been produced in either way.

But cerebral anæmia and apoplectic stupor may also be suddenly produced by emotional causes, such as terror, anger, and despair. These cases also belong to the category of nervous apoplexy. Niemeyer recognizes this relation. While enumerating the causes which produce anæmia of the brain and its membranes, he says: "Cases where, from mental excitement, without lessening of the heart's action, there are paleness of the cheeks and even loss of consciousness and other symptoms of insufficient supply of blood to the brain, seem to indicate that anæmia of the brain may also be caused by abnormal innervation or spasmodic contraction of the [cerebral] arteries." In another place he says: "The symptoms of anæmia of the brain that comes on suddenly, and quickly attains a high grade, differ from those due to one which comes on slowly and is less severe. In the former case the patients become dizzy; every thing appears dark before them; they become insensible to impressions and incapable of movement; their pupils dilate, their respiration becomes slow, and they lose consciousness; they sink to the ground, usually with slight spasms. In most cases the patients come out of this fainting fit in a short time; in other cases, usually termed *apoplexia nervosa*, consciousness does not return; the swoon ends in death." "In anæmia of the brain that comes on slowly, just as in hyperæmia, at first there are usually symptoms of [cerebral] irritation, subsequently those of [cerebral] paralysis." (Vide *Niemeyer's Text-Book of Practical Medicine*, vol. ii. pp. 170–172, 1st Am. ed.)

Dr. Copland has related a case of nervous apoplexy which occurred in his own practice. Cases of apoplexy wherein no morbid appearance was found after death have also been recorded by Willis, Stark, Powell, and Abercrombie; and similar cases have occurred to Morgagni, Tissot, Quarin, Ozanaham, Foderé, and Hildenbrand. The term nervous has been applied to this variety of apoplexy by several eminent authors, particularly Kortum, Zuliani, and Hildenbrand. Apoplectic seizures terminating quickly in death have occasionally been observed in epileptics and maniacs, as recorded by Foderé, Nacquart, Belloc, and Gendrin, without any lesion of the encephalon being manifest. (Vide *Copland on Palsy and Apoplexy*, pp. 111, 112.) Lecat and Weikard attributed the occurrence of nervous apoplexy to spasm of the nerves and vessels of the brain. (*Copland.*) Indeed, it is not difficult to understand that spasmodic contraction of the cerebral arteries, when it occurs on a large scale, is likely to occasion apoplectiform phenomena by inducing cerebral anæmia. Spasmodic contraction of a severe character involving all the branches of the internal carotid would cut off the blood-supply of the corresponding portion of brain almost as effectually as embolism of the internal carotid itself; and spasmodic contraction involving all the arteries of the brain would operate still more powerfully in the same direction. We should, however, state that, in all probability, our predecessors not unfrequently mistook embolic for nervous apoplexy, and sometimes perhaps uræmia for that affection. It is also probable that hereafter, as our knowledge of internal pathology increases, the term nervous apoplexy will become restricted to the cases wherein the apoplectiform phenomena are due to spasmodic contraction of the cerebral arteries, occasioned either by reflex nervous irritation or by powerful mental emotions, as we stated at the beginning of this chapter. The symptoms of nervous apoplexy bear no inconsiderable resemblance to those of epilepsy, provided the convulsive movements belonging to the latter disease are left out of view. In both, the attack begins with the same pallid hue of the countenance, the same bloodless condition of the brain, and the same evidences of general prostration. It is also probable that spasmodic contraction of the cerebral blood-vessels plays an equally important part in the production of both of these disorders.[1]

For an account of the *diagnosis* and *treatment* of *nervous* apoplexy, see *diagnosis* and *treatment* of *apoplexy* in general in Chapter X.

2. ON GOUTY APOPLEXY OR APOPLECTIFORM CEREBRAL GOUT, AND THE FUNCTIONAL DISTURBANCES OF THE BRAIN IN GENERAL WHICH ARE PRODUCED BY THE GOUTY DIATHESIS, OR CEREBRAL GOUT IN THE UNRESTRICTED SENSE OF THE TERM.

The sagacious Heberden remarks: "The gout disposes the subjects of it to apoplexies, either by a general debilitating of the powers of life, or by some affinity between the causes of the two distempers." (Vide *Commentaries on the History and Cause of Diseases*, by W. Heberden, London, 1802.) We have already shown in Chapter III. that apoplexy is not unfrequently connected with the gouty kidney or the gouty diathesis as its cause, and that Drs. Garrod, Gairdner, and others enumerate apoplexy among the gouty affections of the brain. Dr. Tanner, in speaking of the complications which are liable to attend an attack of gout, says it may produce "intense headache, lethargy, and sometimes apoplexy or paralysis." (Vide *Practice of Medicine*, p. 192, Am. ed. 1870.) Dr. Aitken, and modern authors generally, recognize apoplexy as one of the possible consequences of gout.

Dr. Copland has related the following instructive case of apoplectic gout, which occurred in his own practice. He says: "Some years ago I was called to a medical friend in Westminster who, after complaining of symptoms of indigestion, was suddenly seized with complete apoplexy, with [coma and] stertorous breathing, but no paralysis, for which the usual means were promptly and decidedly employed. On the following day a complete attack of gout in both feet took place, with complete disappearance of the cerebral disease. Warmth to the feet, and aperients, were prescribed; but from his eagerness to rid himself of the pain, and to visit his patients, he took, contrary to the advice given him, a large dose of colchicum. A few minutes afterward the gout left his feet and seized his stomach; whence it was with difficulty recalled to the extremities. This was the first time he had ever been seized with gout, and the first part it attacked was the brain in as complete a form of apoplexy as can be conceived." (Vide *Copland on Palsy and Apoplexy*, pp. 200, 201.)

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[1] In epilepsy the condition of the cerebral circulation at the beginning of a fit is very similar to that which obtains in at least some of the cases of nervous apoplexy. There is in both a state of cerebral anæmia which is due to sudden spasm, of a tonic character, of the cerebral blood-vessels. The objective symptoms also are the same in both, excepting the convulsive movements with which epilepsy is usually attended.

Dr. Gairdner mentions the case of a lady, aged somewhat more than 50, who for several years was the victim of misplaced, wandering, or concealed gout. He says: "She often declared to me that she knew she had the gout in her system, struggling for issue. In this state I was summoned one day to see her; she had been struck down with apoplexy. It became necessary now to take more blood. On her recovery she had symptoms of hemiplegia, which, however, entirely disappeared at the end of a week. She now again thought herself safe, and forgetting all rules of diet as before, relapsed into a similar illness, from which she recovered only to fall a victim to ascites, little more than three years from the beginning of her sufferings." (Vide *Dr. William Gairdner's Treatise on Gout*, etc., p. 35. London, 1854.)

Niemeyer states that he has seen cerebral troubles occur in two patients, who also suffered from gout, which he considers to have been due to a circumscribed gouty affection of the meninges: "All other diseases of the brain or its membranes could be excluded with certainty, especially when the symptoms, which appeared very dangerous and excited great anxiety, disappeared in the one case with a copious excretion of urates in the urine; and in the other, with an attack of gouty inflammation of the joints." In another place he says: "Gout in the brain may sometimes present the appearance of apoplexie foudroyante, while in other cases it manifests itself by severe circumscribed headache, dizziness, and vomiting." (Vide *Niemeyer's Text-Book of Practical Medicine*, vol. ii. pp. 501, 502, 1st Am. ed.)

Trousseau relates a very striking example of irregular or masked gout which finally resulted in death from apoplexy. The patient was an English major, who had long been subject to hemicrania or megrim, recurring with a periodicity so well marked every Wednesday that almost the exact hour of seizure was known in advance. Trousseau did not suspect the nature of his disease until a frank attack of acute gout showed itself in his foot. This inflammation subsided under antiphlogistic treatment. From that time the patient lost his former good health. He had a second attack, which was an attack of chronic gout, irregular, moderate, and atonic. Not only was his general health altered, but there was likewise a deplorable corresponding effect produced upon his spirits and mental powers. He lost his mental acumen and habitual gaiety of manner; he became heavy, cross, and tiresome. Ere long, he had a first attack of apoplexy; and two years later, he was carried off by a second attack. Trousseau also mentions a case that he saw with Dr. Chaillou in which apoplectiform phenomena were present; and another instance belonging to the same category that occurred in Dr. Demarquay's practice. This patient was a man who, being attacked in the foot by very acute regular gout, with a view to soothe his intolerable sufferings, applied compresses wet with cold water to the affected part. The pain was almost immediately relieved; but a few hours afterward, Dr. Demarquay was sent for in great haste, and found the patient in a state of apoplectic semi-stupor. He spoke with embarrassed voice, and sputtered out the few words which he attempted to pronounce. Fortunately, sinapisms applied to the feet restored the articular inflammation, and then the cerebral symptoms immediately disappeared.

Trousseau relates another very interesting case of cerebral disturbance of gouty origin, but of a different character. This patient was a man aged 40, who had suffered from gout for many years. The attacks finally became atonic, and he was obliged to keep his room for several months at a time; and even to rest in his arm-chair. The pains with which he was tormented were much less localized, but as he could not, or would not, endure them, he had recourse to opium, the doses of which he rapidly augmented. During the latter years of his miserable existence, this unfortunate man became quite powerless. His temper, naturally headstrong, became still more acrimonious, rendering him insupportable to those around him. Without any apparent provocation, he gave way to veritable paroxysms of rage. At a later date, he fell into a condition resembling dementia. Becoming unable to do any thing for himself, it was necessary to lift him out of bed, dress him, and place him on a seat, where he remained for the day. Bent down, and very different from what he used to be in respect to care of his person, he reminded one of the helpless patients to be seen in lunatic asylums. But in conjunction with this state of brutishness, there was no definite phenomenon indicative of mental alienation. Such was the view taken by a physician well qualified to give an opinion in such a case, who was consulted. There was nothing whatever like delirium; and when the patient was roused from his state of torpor, he always replied with precision to the questions addressed him. He had no symptoms of paralysis. The functions of organic life were performed without perceptible difficulty. The circulation never seemed to be embarrassed; respiration was regular; he retained his appetite; and his digestion continued to be perfect. At last, he was unable to leave his bed; day by day, his torpor increased; and he died in a state of coma. (Vide *Trousseau's Lectures on Clinical Medicine*, vol. iv. pp. 377–386, New Sydenham Soc. translation.)

Dr. Gairdner relates the following case of sudden death occurring in a gouty subject without apparent cause: "A gentleman, aged 63, complained of some disturbance of the stomach, with confined bowels, foul tongue, feverish condition, and dejection of spirits. He sent for a neighboring surgeon, a young man, who had never had charge of his health before. A brisk cathartic of calomel and extract of colocynth was administered to him, which was followed by a dose of senna and salts. Not being relieved the next day, but, on the contrary, feeling somewhat worse, a similar dose was prescribed, which acted with great severity. He was much exhausted by these evacuations, and while sitting on the close-stool, his looks appeared vacant and ghastly. He was quickly removed to his bed, and had no sooner reached it, than he swooned and died, amid the consternation of his friends. In this case, at the autopsy, *all the organs of the body were found healthy.* I had repeatedly attended this gentleman for gout, his only malady. It was always of the atonic, and sometimes of the misplaced kind, attacking the chest and stomach. I had never known him have a very painful fit . . . . . .; he always suffered much from depression of spirits and dyspepsia." (Vide *Dr. William Gairdner's Treatise on Gout*, etc., pp. 45, 46. London, 1854.) This man's death was probably due to the action of the gout-poison upon his nervous system.

Again, Dr. Gairdner says: "If I may trust my own experience, the metastasis of gout more frequently takes place to the head than to any other part. It is usually stated that it shows itself in the common forms of apoplexy and paralysis. I have found these the rarest forms of the disease. I have more commonly seen a kind of stupor, in which the patient preserves his senses of hearing and sight, but loses his consciousness of persons and circumstances, place and time. He knows no one about him, not even his own family; his utterance is imperfect, or altogether lost; he seems like a person entranced; his eyes are vacant and staring; his pulse is full and hard; he understands some of the things said to him, and will do as he is bid, if that which is demanded of him may be easily and quickly done. When asked to do so, he will hold out his hand or show his tongue, but is unable to comprehend any lengthened phrase. He probably hears only one word in a sentence, and does not know any thing or person around him, unless his attention be forcibly called and pointed to the object; yet he smiles stupidly on all, and seems conscious of his own infirmity. But matters do not arrive all at once at this degree of exasperation. Long before the healthy or physiological action of the brain is thus completely overcome, it has been obscured. The patient has suffered from violent and pertinacious headaches. He is observed to be somnolent, especially after repasts. He shows less alacrity of mind, and loses interest in things which formerly occupied him. He is himself conscious of less mental aptitude, and regrets the loss of former vivacity. Such is, according to my observation, the common form of metastasis of gout to the brain, in which it is not difficult to discern the first stage of oppression. If it proceed to complete apoplexy, the symptoms do not vary from those of its ordinary form. When cerebral disturbances occur, it will generally be found that the urinary and hepatic secretions are either totally or partially suppressed. Matters which should be eliminated from the body, thus retained in the system, never fail, even in a moderate degree, to excite great disturbance, of which we shall find ample evidence as we proceed with our inquiry." (Vide *Dr. William Gairdner's Treatise on Gout*, etc., pp. 77–79. London, 1854.)

(*Severe and protracted gout; crural thrombosis; embolism of pulmonary artery, syncope, and sudden death, with symptoms which might be mistaken for those of cerebral gout; no autopsy; case reported by Mr. Paget.*)

"A member of our profession, whom I saw in consultation with Dr. Ferguson and Mr. Morgan, was suffering with a severe and protracted attack of gout, such as he had had more than once before. During its course he had signs of phlebitis in scattered portions of the veins of the right thigh and leg, (having previously had phlebitis three times from accidental causes.) He was sufficiently recovered to be down-stairs and engaged in writing, and thought himself convalescent; but having walked up-stairs to his bedroom, he fell down as if in a deep syncope, and remained nearly an hour, breathing very faintly, scarcely conscious, and with a feeble, fluttering pulse. In a few hours he seemed quite recovered, and next day, and two days later, we could find nothing additionally wrong about him, except a fresh attack of similar phlebitis in the opposite thigh. We examined his chest, and detected only some slight crepitus and faint breathing about the root of one lung. All appeared going on well for three days, and he had no sign or warning of severe illness; but five or six days after the previous fit, as he was sitting on the night-stool, he fell forward, and rapidly died, with a renewal of the signs of syncope and feeble breathing. No examination after death was made; but," says Mr. Paget, "from the likeness of the manner of death to that which I have seen, in cases of ascertained embolism from the systemic veins into the pulmonary artery, I cannot doubt what happened here. It is probable that in the first fit the obstruction of the pulmonary artery was partial; or, that the clot was broken up, and its fragments dispersed; and that in the second, another clot became blocked in the main artery, or was heaped on the adherent fragments of the previous clot." (Vide *St. Bartholomew's Hospital Reports*, vol. ii. 1866, pp. 83–85.) As mentioned above, this case might easily have been mistaken for one of cerebral or apoplectic gout. On close examination, however, some striking points of difference appear. The syncope, the feeble breathing, the fluttering pulse, and the sudden death, were due not to a cerebral but to a pulmonary lesion.

Periodical headaches or megrims are in many cases so evidently manifestations of the gouty diathesis, that megrim and articular gout are observed in the same person, the one subsiding on the appearance of the other; and that megrim is often, also, the only expression of the hereditary tendency in subjects who are the children of gouty parents. (*Trousseau.*)

Vertigo sometimes occurs in consequence of masked gout, as it did in the man of whom Boerhaave's commentator relates, that during two years he was always seized with vertiginous symptoms on attempting to stand up. In vain did the ablest practitioners endeavor to cure him. Quite suddenly he had an attack of gout, of which disease till that time he had given no indication; from that date he was entirely free from distressing vertigo.

Cerebral gout may take the form of epilepsy. Von Swieten mentions the case of a man, his patient, who had violent abdominal pains, accompanied by delirium and general tremor; at a later period he had a severe attack of epilepsy. From the date of that seizure, he had twice a year attacks of regular gout, and was no longer tormented by the nervous symptoms he had previously experienced. The following case is doubtless of a similar nature.

Epilepsy in a Gouty Subject cured by Colchicum.—Dr. Rounel was called to see M—— P——, a bailiff, by his son, who said his father was dying in an epileptic fit. By the time the doctor arrived the fit had passed, and the man was able to tell him that he had suffered from epilepsy for ten years, and had consulted many noted physicians; but in spite of the most faithful trial of their remedies, the paroxysms were constantly becoming more frequent and severe. The various prescriptions (copies of which had been kept) comprised almost all the recognized anti-epileptic drugs. On questioning, it was found that the patient had formerly been a martyr to gout, that the first epileptic fit was immediately preceded by a severe attack of gout, and that he had been free from gout ever since the fits began. He was at once put upon the steady use of colchicum, and after a short time was freed from his epilepsy. (Vide *New Remedies*, January, 1872, pp. 215, 216; also, *Revue de Thèrapeut. Medico-Chirurg.*) In all probability this was a case of epileptiform cerebral gout.

Occasionally, the sensorium is disturbed by the materia morbi of gout. A man complained that his sight was impaired; his eyes, he said, seemed as if covered with a flake of snow. These sensations disappeared, and his vision was restored after an attack of gout in the foot.

Lowness of the spirits or melancholy, and certain delusions called vapors, together with other symptoms of a hypochondriacal or hysterical character, are often due to concealed or masked gout, and disappear at once on the occurrence of a regular paroxysm of that disease; and we believe that insanity itself is occasioned by larvaceous gout much oftener than is generally supposed.

Dr. Gairdner relates two striking cases in which the mind was seriously affected in consequence of concealed gout, and intense hypochondriasis or melancholia was thus produced. Both of them occurred in his own practice. (Vide *Dr. William Gairdner's Treatise on Gout*, etc., pp. 60, 61. London, 1854.)

Again, the most striking among the premonitory symptoms of an attack of gout are certain disturbances of the cerebral functions. The gouty subject, at this period of his attack, complains of weight in the head and inaptitude for every kind of intellectual labor. But the abnormal condition of the brain is indicated principally by a nervous excitability which is often excessive in both regular and irregular attacks of gout, but particularly in the latter. This nervous excitability shows itself by phenomena which vary much in character, according to the individual. There is an undefinable sense of discomfort and mental uneasiness, and curious changes of disposition. Though some persons show an exaltation of their brilliant qualities, this is far from being always the case. Gouty persons generally acquire a morose, susceptible, and irascible temper, formerly foreign to them. This is so usual that it has passed into a proverb among authors on gout. So great, sometimes, is this perversion of disposition, and so constant is it in some persons, that not only do the subjects themselves know that an attack is coming on, from the state of their feelings, but those about them can also foretell the attack from observing these premonitory indications.

Let us briefly enumerate the several forms of cerebral disorder which the materia morbi of gout has been known to occasion. They are a dreadful feeling of heaviness in the head and general uneasiness, with lassitude, and inability to apply the mind to any thing; perversion of temper, obscuration of intellect, lowness of spirits, profound melancholy, hypochondriacal delusions, brutishness, and insanity; intense circumscribed headache or megrim, which is often distinctly paroxysmal in character; impairment of the special senses, such as sight and hearing; general hyperæsthesia, vertigo, and spasmodic attacks of vomiting, with nervous dyspepsia, due to cerebral irritation; paroxysms of epilepsy or eclampsia; stupor, lethargy, and finally all the symptoms of apoplexy in the etymologically correct sense of the term. That this terrible picture of the consequences which may be produced by the action of the gout-poison upon the brain is not overdrawn, the author knows from personal experience and observation, as well as from reading. Some of the above-mentioned phenomena generally present themselves in the forming stage of acute regular gout, others in the retrocedent or wandering variety of the disease, and all of them may be found in the cases of atonic, irregular, anomalous, or masked gout. Some of these phenomena are very often met with, others not so frequently; but it is believed that gouty epilepsy, gouty insanity, and gouty apoplexy occur much oftener than is commonly supposed, and not unfrequently in cases where the real cause of the cerebral symptoms is overlooked.

Nature of Cerebral Gout.—It is for the most part a neurosis. The symptoms are generally the result, not of inflammation, but of functional disturbance of the brain, occasioned by a poisoned state of the circulating blood. It is now generally supposed that the proximate cause of gout is an excess of uric acid, probably in the form of urate of soda, held in solution in the blood. We say that the phenomena of cerebral gout are generally not due to inflammation of the brain or its membranes, because they almost always disappear much too suddenly and completely on the occurrence of arthritic symptoms, to have been produced by any lesion of structure such as inflammatory action would necessarily imply. Oftentimes the relief which the patient experiences as to his head-symptoms on the development of an attack of gout in his foot is so great and occurs so quickly as almost to seem to be due to preternatural rather than to natural causes. Perhaps gouty inflammation of the cerebral meninges sometimes occurs, but if it ever does, it must be very rare. It is probable that in cases of gouty apoplexy hyperæmia of the brain is generally present; but this hyperæmia is always of a passive character and mainly due to sudden dilatation of the cerebral capillaries and stasis of the blood contained in them—the result of sudden paralysis of their walls, or vaso-motor paralysis, as it is sometimes called; and the vaso-motor paralysis itself is occasioned by the action of the gout-poison, the uric acid, upon these branches of the sympathetic nerve. If in cases of apoplectic gout the cerebral hyperæmia does not soon pass away, it is likely to become complicated with œdema of the brain and its membranes.[1] It is not improbable that what we sometimes see occur very suddenly in the skin in cases of atonic gout, namely, purplish-colored congestion with some tumefaction which pits on pressure, but without any pain or tenderness, occurs also in the brain in cases of apoplectiform gout. These phenomena in the skin appear to be due entirely to passive hyperæmia and consequent stagnation of the blood; and they may disappear as suddenly as they came, on the occurrence of gouty symptoms in some other part, or on profuse excretion of the urates. The author has several times experienced this gouty affection of the skin in his own person. It does not appear to be inflammatory in its nature, and doubtless should be considered as a gouty neurosis.

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[1] This cerebral hyperæmia may also induce cerebral hemorrhage in those who are predisposed to its occurrence from aneurismal dilatation or atrophy of the cerebral capillaries. "Sir C. Scudamore mentions that Dr. Parry met with two instances of extravasation in the brain in the same winter, after repelling gout from the extremities by immersing them in cold water." (Vide *Art. Gout (irregular) in Copland's Dictionary of Medicine.*)

Treatment of Cerebral Gout.—We should be careful to avoid the use of lowering remedies, and especially the abstraction of blood, in treating the cases which belong to this category. If retrocession from some joint has occurred, we should endeavor to recall it by the application of rubefacients, such as sinapisms, etc. In bad cases, and especially in the apoplectiform ones, the judicious administration of ether, musk, alcohol, and other stimulants has often been found beneficial; and perhaps they do good by overcoming the vaso-motor paralysis of the cerebral blood-vessels. But general rules cannot be laid down for the treatment of anomalous internal gout. In each case the symptomatic indications should be carefully followed. The application of cold to the head with the ice-bag or frozen cloths often proves singularly useful for the hyperæsthesia and wakefulness on the one hand, and for the lethargy and apoplectic stupor of cerebral gout on the other, especially when the pulse is strong and the pupils contracted, since each of these morbid states depends upon cerebral hyperæmia as its cause, and they differ from each other mainly in respect to the degree of hyperæmia which corresponds to each of them. The good effect of cold to the head is promoted by the simultaneous use of hot mustard-water pediluvia, and sinapisms applied to the epigastrium. The hyperæsthesia and wakefulness is also benefited by the administration of bromide of potassium in doses of from ten to twenty grains twice a day. The lowness of spirits and hypochondriacal symptoms are often considerably relieved by the use of bromide of potassium and the extract of cannabis indica, gr. ½ of the latter in the form of pill twice a day. Guaiacum also has sometimes proved singularly useful in my hands for relieving the dulness of intellect, or mental cloudiness, and the depression of spirits which attend this disease. Gouty headache often requires the employment of colchicum, but we should generally avoid the use of this drug in cases of cerebral and the other atonic forms of this disease unless other remedies have failed. Spasmodic vomiting, the result of cerebral irritation, often proves very distressing in cases of anomalous gout. We have obtained most relief for such cases by administering sulphate of quinine in five-grain doses, and oxalate of cerium in two-grain doses, three times a day. We have also found the sulphur-water of Richfield Springs very useful for the cerebral and other forms of this disease. It should be taken internally, and employed externally in the form of hot baths. Gouty epilepsy requires the administration of bromide of potassium or sodium. At the same time the rules with regard to diet and exercise which are usually laid down by systematic writers on the gouty diathesis should be scrupulously carried out in each of these forms of cerebral disturbance. It does not seem to be necessary to reproduce them here. We must not forget to state that the use of opium or morphia should generally be avoided in cases of cerebral gout, for it constipates the bowels, deranges the stomach, and increases the tendency to hypochondriasis. When hypnotics are required in such cases, as they often are, we should administer bromide of potassium, cannabis indica, or hyoscyamus. In my own experience, a combination of them (potass. bromid., grs. x.; ext. cannabis ind., gr. ½; do. hyoscyami, gr. j.) has oftentimes procured several hours of natural sleep for such a patient without any deleterious consequences. We should also mention that lemon-juice taken freely in the form of lemonade generally proves useful in cerebral gout. The carbonate of lithia has, in my experience, promoted very much the excretion of uric acid with the urine in such cases.

But the victim of the gouty diathesis should always bear in mind that there is no other morbid state of the body in which scrupulous attention to food and drink and exercise is more important than in this. If he is the victim of hereditary gout, he should be taught to know that he cannot make his condition in life even tolerable unless he practises much self-denial in eating and drinking, at the same time leading a regular life in every respect, avoiding over-fatigue, whether from labor or the pursuit of pleasure, and taking much exercise in the open air.

3. On Rheumatic Apoplexy, and the other Forms of Cerebral Rheumatism.

Recently, the subject of cerebral rheumatism has attracted much more attention than that of cerebral gout, but, probably, the former is not any more important than the latter, if, indeed, it is as much so. Rheumatism generally runs its course without exciting much if any cerebral disorder; occasionally, however, the functions of the brain are greatly disturbed by it, and sometimes death is suddenly produced in that way. In the spring of 1860, the author treated a patient for acute rheumatism whose sudden and unexpected death from cerebral disturbance made a most profound impression on him. The following is a brief account drawn up from memory:—

Case XLV.

Acute multi-articular rheumatism ; sudden death with apoplectiform phenomena.

Mrs. H——, aged about 50, wife of a farmer in good circumstances, mother of a large family, exemplary in all her habits, body rather fleshy, health generally good, and not liable to any hereditary disease, was attacked with acute rheumatism in the fore part of May, and I was called to attend her some two or three days afterward. She then had a good deal of fever, full hard pulse, profuse acid perspiration, loaded tongue, and several joints of both the upper and lower extremities were simultaneously affected, but there was no cardiac lesion. She was put on a saline plan of treatment similar to that recommended by Dr. Fuller, namely, she took sal Rochelle, grs. xxx.; soda bicarb., potass. bicarb. āā. grs. x., in solution and well diluted, every four or six hours, according to the effect on her bowels, with pulvis Doveri grs. x. at bedtime, as she complained of having much pain in the affected joints; the Dover's powder was repeated if necessary in order to procure sleep; the affected joints themselves were wrapped up in cotton-wool covered over with oiled silk.

Under this treatment her case seemed to be progressing slowly but, on the whole, favorably toward recovery. The fever had abated somewhat, the joints were rather less painful, her heart remained free from abnormal murmurs, when early one morning, about four or five days after I was first called, or about a week from the beginning of the attack, I was summoned in great haste to visit her, with the message that they thought her dying. I found her lying in a state of stupor or coma, of so deep a character that she could not be rorsed from it; her breathing was somewhat labored and snoring, her skin hot and moist, her pulse rather frequent and feeble, and the heart-sounds rather weak, but otherwise normal. The heart was examined with extreme care. I learned that she had been restless and slightly delirious during the night, her mind was perfectly clear at my visit in the morning, and that she had sunk into an unconscious state just before daylight. She had not had any sudden attack of dyspnœa, nor, indeed, any difficulty of breathing whatever, nor any convulsions, nor any symptoms of paralysis. The coma did not appear to have come on so suddenly as to constitute a fit of apoplexy in the proper sense of the term, although it had been very quickly developed. The affected joints still continued swollen. We endeavored to relieve her brain by putting cold compresses on her head; by applying counter-irritants to the nape of her neck, to the epigastrium, and to the extremities; and by administering diffusible stimulants and digitalis internally, but without deriving any benefit from them. Her pulse gradually became weaker and more frequent, she finally got rattles in her throat, and died comatose about 4 o'clock P.M. I observed that as she grew worse her bodily temperature, as measured by my sense of touch, seemed to increase in a decided manner, instead of diminishing, and that when death occurred her skin felt very hot, thus reminding me strongly of some fatal cases of sunstroke which I had seen. No autopsy was allowed; but it is certain that the head-symptoms were not due to embolism of the cerebral arteries nor to embolism of the pulmonary artery, and it is probable that they did not have their origin in any form of intra-cranial inflammation.

Trousseau has related a case in which death very suddenly occurred from cerebral rheumatism. The patient was a remarkably robust-looking man who was undergoing his fourth attack of acute multi-articular rheumatism. He appeared to be doing well. The clinical assistant observed no unusual symptom at his evening visit; the pain in the joints had diminished, and the patient was very much pleased with his condition. "An hour later, however, he complained of not being able to see, and shortly afterward began to vociferate; he cried out 'Thief!' rushed out of bed, and fell down. On being put back to bed by two attendants, he struggled with them, exhibiting considerable strength, and then, dropping back, died. All this took place in less than a quarter of an hour." On post-mortem examination, the brain was found to present a remarkably healthy appearance. The pia mater exhibited a moderate amount of congestion; but the meninges did not exhibit any changes of an inflammatory nature whatever. The cerebral symptoms, therefore, were not accounted for by the autopsy. (Vide *Lectures on Clinical Medicine*, vol. i. pp. 513–515, New Sydenham Soc. translation.)

Dr. Weber has very carefully reported to the Clinical Society of London two cases of sudden death occurring in the course of acute rheumatism, and due apparently to paralysis of the functions of the brain. As the cerebral symptoms presented themselves, the temperature rapidly rose in both instances, just as it did in the case which we have related. The first patient, a man, aged 45, had the ordinary symptoms of acute rheumatism till the twelfth day, when signs of endocarditis occurred, with great restlessness, deafness, noises in the head, excessive urination; next day, violent delirium and vomiting, rapidly followed by coma and rise of temperature to 109.5°, with intense fever, appeared. Death occurred five hours from the beginning of delirium, and the body rapidly decomposed. The second patient, a young man, aged 25, had seemingly an ordinary attack of acute rheumatism, but with great depression of spirits. On the twelfth day, symptoms of endocarditis occurred, with deafness, increased urination and action of the bowels. Next day, violent delirium appeared, soon followed by coma and very high temperature; death occurred three hours from the beginning of delirium. Cadaver rapidly decomposed; the blood was found in a fluid state, and ecchymosed spots beneath pericardium, pleura, in tissue of lungs, also on liver and kidneys. The onset of fatal symptoms and the termination were remarkably alike, in both these patients; both were highly nervous persons, and had lately been subject to anxiety. In both, no warning was given by the early symptoms of impending danger; the cerebral symptoms were developed with great rapidity, and soon terminated in death.

Dr. Murchison has also reported to the Clinical Society of London two cases belonging to the same category. In the first case, delirium was the first serious symptom, and occurred upon the night of the fourteenth day; it passed by degrees into stupor, and death occurred in less than two days from the commencement of the head-symptoms. The *post-mortem* phenomena were early decomposition of body, blood dark and fluid, spleen large and soft, kidneys enlarged and congested, their tubes gorged with granular epithelium. Nothing wrong in brain, except some congestion of veins and sinuses. Slight signs of recent pericarditis; muscular tissue of heart pale and fatty. The second case was a married woman, aged 26, ill-fed, and having suckled a child for three months. This was her second attack of rheumatism. On the night of the sixth day, delirium occurred; for a short time next day the temperature was 104½°, and there was no delirium, but there had been no sleep. On the following night, after an hour's sleep obtained by opium, the delirium returned and passed into coma; death occurred early the next morning. The autopsy revealed almost exactly the same morbid appearances as were found in the last case, with the addition of waxy changes in the recti muscles of the abdomen.

Dr. Burdon-Sanderson has related another case of the same sort. The patient was an unmarried woman, aged 45, of neuralgic temperament. It was her first attack of rheumatism. The first serious symptoms appeared on the twelfth day of illness, and five days after the first symptoms of rheumatism. The breathing then rose to forty-six, with sudden inspirations; there was also a tendency to stupor. The lungs were congested at the lower part behind. Next day the pulse was 120, the breathing 56, and the rheumatic symptoms were but slight. The following day drowsiness still continued, and in the afternoon the temperature was found to be 109.4°; the suddenness of inspiration was more marked; expiration was also sudden and stridulous, and the pause prolonged. An observation of the pulse under a spring-pressure of 120 grammes showed a pulse-curve of moderate size, with no secondary markings, and much influenced by the breathing. The temperature was taken again in one hour and a half, and found to be 110.2°: shortly afterward she expired. For some time before death the eyes were closed, the pupils contracted, the iris had a tremulous movement and was unaffected by light. On *dissection*, slight signs of pericarditis were found, and changes of the blood and in the viscera, similar to those noticed in the preceding cases.

Dr. Weber expressed the opinion, that in this class of cases the functions of the brain, after a short stage of excitement, become paralyzed, and that the symptoms of intense fever are not the cause but the effect of this paralysis. He compared them with certain cases of other fatal diseases of the nervous system, such as tetanus, observed by Wunderlich, in which there was excessive rise of temperature, and pointed out the resemblance to the phenomena of sunstroke. He also referred to the remarkable experiments of Tscheschechim, who found that, after section of the pons at its junction with the medulla, the contractions of the heart and the respirations were accelerated and the temperature raised. He suggested that all cases of acute rheumatism with more than usual restlessness should be watched with especial care. If the symptoms of danger were early noted, good might be hoped for from the employment of cold affusion or other means of lowering the temperature, combined, perhaps, with large doses of quinine. (Vide *New Sydenham Soc. Retrospect*, 1867–8, pp. 40–42; also *Lancet*, February 1st, 1868, and *Clinical Soc. Transact.*, 1868.)

Dr. Murchison has reported another fatal case of acute rheumatism. It occurred in a carman, aged 26, in whom the temperature rose to 109.5° twenty minutes before death. (Vide *op. cit.* 1869–1870, p. 99; also *Lancet*, 1870, i. 724.)

Dr. Wilson Fox has recorded the case of a housemaid, aged 30, who suffered from acute rheumatism with pericardial effusion, and whose temperature rose to 107.8° at midnight of the second day before death. She was violently delirious at the time. A full bleeding was tried without any good effect, the temperature rising in the next hour to 108.9°. She was then placed in a cold bath (60°) from time to time, and the temperature fell; but, unfortunately, she died about thirty-six hours after the commencement of this treatment. Dr. Fox thinks, however, that the protraction of life for so long a time after the temperature had reached so high a point was due to the cold bath. (Vide *New Sydenham Soc. Retrospect*, 1869–70, p. 99; also *Lancet*, 1870, ii. 7.) This case, as far as it goes, supports the views of Dr. Weber with regard to the probable value of cold affusion in cases of cerebral rheumatism where sudden and great elevation of temperature occurs.

Dr. T. Clifford Allbutt states that he had seen eleven cases of cerebral rheumatism with only one recovery, (and in that case the symptoms were never violent,) prior to last September. Then a very violent case was saved solely, as it seemed, by the abstraction of heat from the patient by means of bottles of ice applied to his body, and ice applied freely to his head, and allowed to drip over his shoulders. This case is related in the *Lancet* of December 23d, 1871. (Vide *New Remedies*, April, 1872, p. 295.)

The foregoing cases of cerebral rheumatism were *apoplectiform*, and not genuinely apoplectic in character. The loss of consciousness and coma did not occur suddenly enough for that. But, when rheumatism occasions apoplectic stupor or a stroke of apoplexy, properly so called, it generally does it by inducing cerebral embolism, a pretty full account of which was given in the last chapter. In such cases the embolia that plug up the cerebral arteries have their origin in and migrate from the left side of the heart. They may consist of the coagulated blood which results from cardiac thrombosis, or of concretions of fibrin which have formed on the inflamed valves and endocardium, or of fragments of the valves themselves when they chance to be the seat of ulceration. But rheumatism may also produce sudden unconsciousness and speedy death by causing the pulmonary artery to become plugged up with a thrombus which has migrated from the right side of the heart or from the great veins connected with the right auricle. Such cases are usually attended with great dyspnœa, but the cerebral symptoms which they present are of a secondary character, and, therefore, they can scarcely be considered as instances of rheumatic apoplexy. We repeat that rheumatic apoplexy is usually a form of cerebral embolism.

In the cases of apoplectiform cerebral rheumatism which we have related, the autopsies did not reveal any lesion of the brain itself or of the membranes adequate to account for the great disturbance of the cerebral functions. In none of them was any thing wrong found in the brain, except some congestion of the veins and sinuses. There was no exudation of lymph nor inflammatory changes of any description. Hence, it is generally held that in cases of this description the cerebral phenomena are due to the action of the rheumatic poison contained in the blood upon the brain-substance in some way not yet explained. But cerebral rheumatism presents itself only in such persons as are already predisposed to its occurrence. It does not occur in all alike. It appears only in those who have inherited or acquired a peculiarly excitable condition of the brain. For example, Trousseau's case, related above, occurred in the person of a hard drinker; that is, in one whose nervous system had acquired a peculiar susceptibility to morbid impressions from the prolonged abuse of alcoholic stimulants. Dr. Weber's cases, also related above, both occurred in highly nervous persons who had recently been subject to much anxiety. One of Dr. Murchison's cases was an ill-fed woman, who had suckled a child for three months. In his other case, no statement is made as to the point in question. Dr. Burdon-Sanderson's case occurred in an unmarried woman of neuralgic temperament. Our own patient was much shaken in her nervous system by the cares and anxieties incident to the charge of a large family of children, who were mainly dependent on her for counsel and assistance. But that excitable condition of the brain which is necessary to the development of cerebral disturbance in those who have articular rheumatism may also be inherited. Hence it is not unusual to find that the ancestors or the brothers and sisters of those who have cerebral rheumatism have been insane, or afflicted with some other important disease of the nervous system. Trousseau has presented several instances of this sort; and in cases of cerebral rheumatism, generally, the rheumatic poison is only the lighted match which explodes the mine that is already charged; for clinical observation has abundantly shown that articular rheumatism of itself exhibits no great tendency to develop cerebral phenomena. In order, then, for cerebral rheumatism to occur, the operation of something more than articular rheumatism is required, namely: there must be an abnormally susceptible condition of the brain itself already present. This much now concerning the apoplectic and apoplectiform cases of cerebral rheumatism.

2. We must next speak of the *paralytic variety* of this disorder. We have already shown that hemiplegia is often produced by cerebral hemorrhage and cerebral embolism, sometimes by what appears to be cerebral congestion, and occasionally by reflex irritation. Yet cases do occur in which, during the course of acute articular rheumatism, a transient hemiplegia shows itself that must be referred to the rheumatism as its cause. Trousseau has related the history of a young girl who was admitted into the hospital, suffering from intense fever, excessive rachialgia like that which announces variola, and from paraplegia. For three days he looked out for the eruption of variola; on the fourth day he had her cupped, when all symptoms of paraplegia disappeared, but amaurosis and hemiplegia immediately occurred. A few leeches were applied behind the ears, and two days afterward, pain in some of the joints was complained of, whereupon the amaurosis and hemiplegia disappeared. The manner in which the paralysis alternated with the other symptoms showed that it was due to the rheumatic diathesis. Trousseau has related another case of similar purport. A hospital nurse was attacked with rheumatism in her left wrist-joint, which caused marked swelling and redness of the skin, and she suffered from it a long time. She was then suddenly seized with vertigo and a sensation of weight in the head, and her limbs became so *paralyzed* that she was unable to work. Her complaint resisted treatment for a long time, and it was only after the lapse of fifteen months that she was able to resume her duties. Trousseau remarks that "in this case rheu-

matic arthritis preceded the symptoms of paralysis, and thus accounted for their production. Should any doubt remain, an analysis of the phenomena would remove it at once, for the symptoms of paralysis and those of articular rheumatism proper will be seen to alternate in the most significant manner. At one time cephalalgia and sensorial disturbances are present; at another, spinal pain and weakness of the lower limbs; sometimes, again, the cerebral and spinal symptoms are replaced by painful swellings of the joints." Trousseau has related a third case. It also occurred in a woman. "She first had an attack of acute articular rheumatism, which seized on the wrists, and then attacked the head, producing stupor for a day or two. The spinal cord was next attacked, and paraplegia followed. For the space of four months, this poor woman thus presented mobile symptoms, suddenly shifting from one organ to another, from the brain to the spinal cord, and from the cord to some part of the limbs." (Vide *Lectures on Clinical Medicine*, vol. i. pp. 521–525, New Sydenham Soc. translation.) In another place, Trousseau mentions that he lately had under his care a man who complained of very intense pain along the spine, and at the same time was paraplegic. At first he thought the patient was going to have small-pox, but no eruption appearing at the usual time, he made a more careful examination and suspected acute myelitis; a few days afterward, however, the man was seized with articular rheumatism. The inference is that the rheumatism affected the spinal cord before it attacked the joints in this case. (*Ibidem*, p. 534.)

We also infer, from the shifting character of the symptoms which predominated in the cases just related, that the paralysis was not due to the occurrence of effusion in the nervous centres. The metamorphoses in the phenomena occurred far too rapidly for that to have been the case. The paralysis resulted from functional disturbances, and not from organic lesions.

But, it should not be forgotten that when hemiplegia occurs in the course of a rheumatic attack, it is not unfrequently due to cerebral embolism. One of the so-called vegetations formed on the mitral or aortic valves, as an event of endocarditis, being washed off by the blood-stream, migrates to the head, and, by blocking up a cerebral artery, gives rise to sudden asphyxia of the brain from arresting its supply of arterial blood. The phenomena and other consequences of this accident have been sufficiently discussed in the preceding chapter.

3. *Mental Alienation* or *Insanity* constitutes a *third* variety of cerebral rheumatism. Griesinger speaks of it as follows: "The severe cerebral disturbance, lasting for months or more, caused by articular rheumatism, appears as insanity without fever, characterized by depression, or as pro-

nounced melancholy, with stupor. It may be followed, or may alternate, with states of excitement; sometimes this disturbance is accompanied by convulsive choreic movements; the prognosis is, on the whole, favorable; recovery seems to result most rapidly and certainly where, after a time, during the cerebral disturbance, the joints are again attacked with acute rheumatism." (Vide *Niemeyer's Text-Book of Practical Medicine*, vol. ii. p. 482, 1st Am. ed.)

Sander has reported five cases of mental disease which resulted from rheumatism, with the following conclusions: (1.) That severe mental disorder may occur, not only during the persistence of acute rheumatism in the joints, but be prolonged for a month or upward after it has ceased. (2.) This mental lesion manifests itself without fever, usually with the character of depression, and often as decided melancholia with stupor. A state of excitement may follow, or be intercurrent with the melancholia. (3.) Now and then, convulsive or choreic movements complicate the mental disturbance. (4.) The prognosis is very favorable; and recovery, so far as the few recorded cases go to show, comes most rapidly and surely when a fresh attack of rheumatism supervenes in the course of the cerebral affection. It may generally be assumed that the brain-affection and the rheumatism stand more closely related than do the chronic cerebral disorders consequent upon other acute diseases; as, for instance, typhus, where anæmia of the brain, or some other general cause, may be assigned as the basis of the psychosis. The question for solution is whether this association of rheumatism and insanity is attributable to the rheumatic poison acting upon the cerebrum and producing a form of rheumatic meningitis. (Vide *New Sydenham Soc. Year-Book* for 1864, p. 107.) Trousseau has related two cases (one his own, and the other Dr. Mesnet's) in which chronic delirium was produced by cerebral rheumatism. Sulphate of quinine was prescribed for the latter in gradually increasing doses, and remarkable improvement followed. (Vide *Lectures on Clinical Medicine*, vol. i. p. 526, New Sydenham Soc. translation.)

4. A *fourth* variety of cerebral rheumatism is characterized by the occurrence of *spasms* or *convulsions*, which are not due to inflammation of the meninges of the brain. The following are examples of it:—Dr. Bright, in his essay "On Spasmodic Diseases accompanying Affections of the Pericardium," published in the *Medico-Chir. Transac.*, vol. xxii., reports the case of a young man who had been suffering from acute rheumatism for six days, when spasmodic symptoms appeared, increased rapidly in severity, and were shortly accompanied by delirium. This ultimately became so violent that he had to be put under restraint. He died at the expiration of three weeks, and, on dissection, the brain was found perfectly healthy,

and the pericardium and endocardium presented unequivocal signs of recent active inflammation. Dr. Todd, in his "Lectures on Delirium and Coma," delivered before the College of Physicians in 1850, gives the case of a young woman who, after suffering for some days from rheumatic fever, was seized with delirium, and in a few hours afterward had a convulsive fit, succeeded by coma and death; yet the closest examination of the parts after death, while it disclosed extensive inflammation of the pericardium, could not detect a trace of inflammation of the brain, which, together with its membranes, was unusually pale. In two cases of acute articular rheumatism, complicated, in the one, with occasional delirium and choreic twitching of the voluntary muscles for three days, and in the other, with occasional delirium and slight opisthotonos for two days, mentioned by Dr. Fuller in his work on Rheumatism, etc., pp. 204, 206, an examination of the body after death disclosed no inflammation of the brain or its membranes. (Vide *Dr. Bazire's Notes to Trousseau's Lectures on Clinical Medicine*, vol. i. p. 530, New Sydenham Soc. translation, from which the above-mentioned cases are quoted, with but slight alterations.) These were all acute cases of cerebral rheumatism. In two of them the convulsions were probably epileptiform or eclampsic in character, and in one they were tetanic, as they took the form of opisthotonos. It was observed in Dr. Todd's case that the brain and its membranes were unusually pale or exsanguinated. This is what we would expect to find if the fatal coma were due to narrowing of the cerebro-meningeal blood-vessels, from spasmodic contraction of their walls in consequence of irritation of their vasomotor nerves.

Dr. Fuller has related a very instructive case of this variety of cerebral rheumatism. The subject was Harriet Keating, an overworked house-servant, aged 19, who recovered after she had had furious delirium and violent tetanic spasms, whilst suffering from rheumatic fever complicated with pericarditis. She got very low, and did not begin to mend until she was allowed strong beef-tea, together with Hoffman's ether and morphia in full doses. Her recovery became complete on continuing the use of nutrients, tonics, stimulants, and sedatives. (Vide *Fuller on Rheumatism*, etc., pp. 296–300, 3d ed.) Dr. Fuller mentions another case in which there was violent delirium and incessant convulsions for nine days, and still the patient recovered. The patient was a female aged 19. "The treatment throughout consisted of bleeding, general and local, repeated blistering, and mercurialization." The occurrence of ptyalism was attended with great improvement. (*Ibidem*, pp. 311, 312.)

5. The *choreic* constitutes a *fifth* variety of cerebral rheumatism. That chorea is intimately connected with articular rheumatism has long

been known. Dr. Bright states that in the very excellent *Syllabus, or Outlines of Lectures on the Practice of Medicine*, in the edition of 1802, he finds rheumatism distinctly mentioned as one of the exciting causes of chorea; and in later editions, as in that of 1820, it is stated that "chorea sometimes alternates with rheumatism." (Vide *Dr. Bazire's Notes to Trousseau's Lectures on Clinical Medicine*, vol. i. pp. 528, 529, New Sydenham Soc. translation.) Dr. Sée asserts that a child who has one or more attacks of acute articular rheumatism will sooner or later have chorea. Conversely, Trousseau declares that a child who has one or more attacks of chorea will sooner or later have rheumatism. Trousseau relates the case of a girl who was caught round the body by a man on a dark staircase, and shortly afterward became affected with unilateral chorea. This was soon replaced by acute articular rheumatism, and when the rheumatism got well, the chorea returned. Trousseau also relates a case of acute articular rheumatism in the course of which chorea manifested itself with violent symptoms. This patient was a young girl whom he saw in consultation. She had been suffering from acute articular rheumatism for the last ten days, and for two days previous to his visit, violent chorea had set in, with delirium, inability to eat or drink, and constant retching or vomiting. She died from the violence of the chorea. (Vide *Lectures on Clinical Medicine*, vol. i. pp. 527–529, New Sydenham Soc. translation.) From the foregoing cases and statements, we infer that acute articular rheumatism is not unfrequently transformed into St. Vitus's dance, that is, into a cerebral affection, which is sometimes grave and sometimes mild; and that a choreic form of rheumatism must be admitted. M. Thore has related a case of choreic cerebral rheumatism which possesses great interest. The patient was a young lady who experienced an attack of acute articular rheumatism, with pleurisy and endocarditis. Coincidentally with the subsidence of the pains she began (twenty-four days from the commencement of her illness) to be affected with choreic movements, chiefly of the left arm and of the face. Two days later, alarming hallucinations of sight, hearing, and feeling occurred; for two or three days these were very distressing. The mental symptoms and the chorea diminished simultaneously, but the patient remained abstracted and timid for some days. The chorea disappeared about nineteen days after its first occurrence, and only a little weakness remained; a week later the patient was quite well. (Vide *New Sydenham Soc. Retrospect*, 1865–6, p. 84.)

Furthermore, experience has shown that chronic endocarditis is pretty frequently met with in children having St. Vitus's dance, and that pericarditis is of equally common occurrence in both children and adults who are subject to this disease. Now, endocarditis and pericarditis are both intimately related to the rheumatic diathesis and to acute articular rheumatism;

and thus again we find some proof that chorea may be rheumatic in its origin.

Henoch found rheumatism and cardiac disease in five out of fifteen cases of chorea occurring in children. He also found arsenic the most serviceable remedy. H. Roger says there is an interdependence between chorea, rheumatism, and cardiac disease in children. He admits a cardiac chorea, but says the heart-disease and the chorea may start together from the same cause, as is sometimes the case with rheumatism and carditis. His cases prove that the heart-disease often persists after the cure of the chorea. He further shows that the chorea may precede the occurrence of the cardiac lesion. (Vide *New Sydenham Soc. Retrospect*, 1867–8, p. 431.)

Dr. Ferber, after a careful perusal of the literature on the subject, especially of the works of Roger and Tüngel, remarks that there is a want of truth in the statement that chorea, as a complication of acute rheumatism, is confined to childhood, and cerebral disturbance to adults. He concludes (1) that both may occur in connection with acute rheumatism at any period of life; they are found most frequently, however, in children. (2) In children, the symptoms of disturbance in the motor system predominate; in adults, those of disturbance in the emotional or psychical system. In children, chorea occurs mostly without emotional or psychical mischief; while in adults mental disturbance more frequently presents itself without chorea. The symptoms of both chorea and mental disturbance sometimes make their appearance in the acute stage of rheumatism, and frequently are in direct relation with the onset and remission of the articular affection; in children especially the former; in adults, the latter. But generally the nervous symptoms make their first appearance after the rheumatism has abated. (3) In no case does delirium show itself as a premonitory symptom of rheumatism, while in children chorea has frequently been observed to do this by Roger and Henoch. He gives a case in which repeated attacks of rheumatism had occurred in a boy, aged 8, since his second year, the last being attended with disturbance of the sensorium, chorea, and contraction of the lower extremities, the latter occurring without any rheumatic affection of the hip-joint. Dr. Ferber thinks this contraction was due to mischief in the central nervous apparatus, perhaps a slight cerebro-spinal meningitis. (Vide *New Sydenham Soc. Retrospect*, 1869–70, pp. 98, 99.)

But rheumatic chorea is not characterized by any structural lesion of the brain or spinal cord that we are acquainted with, any more than idiopathic chorea is. Trousseau says: "If you examine the bodies of those who have died from the violence of the chorea, and make most careful search in the brain and spinal cord, you will find neither intense congestion, nor softening, nor extravasation; in a word, you will detect no serious lesion, nothing which can adequately explain the symptoms noted during life. Up to the present time, at least, no such lesion has been discovered." (*Ibidem*, p. 532.)

6. Finally, there is a *sixth* variety of cerebral rheumatism, namely, the *meningitic*. It consists of rheumatic inflammation of the cerebral meninges, and is of comparatively rare occurrence. Niemeyer says: "In a few instances the severe brain-symptoms depend on inflammation of the meninges, that is, on changes analogous to those in the joints. I myself have seen only one case where the moderate increase of bodily temperature, the delirium in the first stage, and the subsequent coma, together with excessive retardation of the pulse and repeated vomiting, left no doubt as to the nature of the disease. The case ended in recovery, so that the diagnosis was not confirmed by autopsy; but in a dissertation written by Dr. Flamm, under the direction of my colleague Köhler, on 'Meningeal Symptoms in Acute Rheumatism,' cases are reported where post-mortem examination showed the presence of inflammatory disease of the meninges." (Vide *Niemeyer's Text-Book of Practical Medicine*, vol. ii. p. 482, Am. ed.)

Trousseau mentions a case, recorded by Dr. Marrotte, in which considerable effusion rapidly took place, and symptoms showed themselves indicative of cerebral compression, such as hebetude, dilatation of the pupils, and coma. It was, then, a case of true acute hydrocephalus. (Vide *op. cit.* p. 527.)

In some rare instances, however, rheumatic meningitis may be attended with the formation of pus. Thus, in a female patient, who died under Sir T. Watson's care in the Middlesex Hospital, after symptoms of cerebral inflammation supervening upon acute rheumatism, undoubted pus was found smeared over the hemispheres. The history of a similar case is related by Dr. Fyfe, of Newcastle. A man, aged 36, after suffering for some days with acute rheumatism, was seized with delirium and unequivocal symptoms of cerebral inflammation. Life continued for five days longer, and throughout that period there was either muttering delirium or a state of perfect coma. On the fifth day, at noon, he died; and dissection showed the membranes of the brain covered with lymph and pus, the vascularity of the brain enormously increased, and the lateral ventricles distended with serum. Dr. Fuller mentions an analogous case which came under his observation in St. George's Hospital. A man was admitted, under the care of Dr. Seymour, with his joints inflamed and swollen. One day his knees, which had been greatly swollen, became very much smaller and flaccid, and, coincidently with the subsidence of the swelling, he complained of pain in the head, became paralyzed on one side, and expired in thirty-six hours. On opening his head, a large quantity of greenish-looking purulent matter was found smeared over the greater part of the surface of the left hemisphere, and there was considerable effusion into the ventricles. Dr. Fuller believes that in all such cases the meningeal inflammation is not the result of simple extension of the disease, but of concentration of the rheumatic virus upon the brain in consequence of the sudden subsidence of the articular inflammation. (Vide *Fuller on Rheumatism*, etc., pp. 302, 303, 3d ed.)

But the occurrence of well-marked head-symptoms in the course of a rheumatic fever must not always make us think that meningitis has occurred. Sir T. Watson has recorded four cases of acute articular rheumatism, complicated with carditis and head-symptoms, the latter suggestive of inflammation of the membranes of the brain, which was disproved, however, by post-mortem examination. In three of these cases some serous fluid was found in the meshes of the pia mater and in the lateral ventricles. Dr. Latham has related the following remarkable case in his "Clinical Lectures:" "One of the children of Christ's Hospital had, in the opinion of all who saw him, the severest inflammation of the brain. The attack was sudden, with great heat, and frequency of pulse. He had delirium and convulsions, and pointed to the forehead as the seat of his pain. In three days he died, and, upon dissection, not a vestige of disease was found within the cranium; but the heart was exclusively the seat of the disease, and no other part of the body exhibited the slightest morbid appearance. The disease of the heart was not confined to its investing membrane; it was the most intense inflammation, pervading the pericardium and the muscular substance." In a second instance, mentioned by Dr. Latham, "the whole force of the treatment was directed to the head, from a belief that the brain was inflamed. Upon dissection, the brain and its coverings were found in a perfectly healthy and natural state; and the pericardium, toward which during life there was no symptom to direct the slightest suspicion of disease, exhibited the unequivocal marks of acute and recent inflammation." Dr. G. Burrows, in his essay "On Disorders of the Cerebral Circulation," at p. 188, mentions the case of a shop-boy who died, after seven days' illness, in a state of restlessness and delirium. On dissection, the brain and its membranes were found normal, while the pericardium was found covered over with fresh lymph; and "upon the anterior surface of the left ventricle of the heart there was a white spot, about a quarter of an inch in diameter, which appeared to be formed by concrete pus." Again, in a patient who died lately in the Middlesex Hospital, under the care of Dr. Murchison, of rheumatic fever complicated with delirium and marked head-symptoms, no alteration of the brain or meninges was found after death. (Vide *Dr. Bazire's Notes to Trousseau's Lectures on Clinical*

Medicine, vol. i. pp. 529, 530, New Sydenham Soc. translation.) To the foregoing we might add many other cases of similar import, if it were necessary to do so. Trousseau has fully recognized the difficulty which often attends diagnosing the meningitic variety of cerebral rheumatism, and broadly asserts that it is "an anatomical, not a clinical, form of the disease." However, rheumatic meningitis may sometimes be recognized during life by bearing in mind the symptoms which Niemeyer found in his case, namely, temperature of patient only moderately increased, delirium in first stage followed by hebetude and coma, together with excessive retardation of the pulse and repeated vomiting. Dilatation of the pupils occurring in connection with other symptoms of acute hydrocephalus, may prove of some value as a sign of the copious and rapid effusion of serum. Hemiplegia developed, not suddenly, but gradually or by degrees, in a case of cerebral rheumatism, along with phenomena of meningitis, as it was in Dr. Fuller's case mentioned above, will render it probable that abundant exudation of the products of inflammatory action has occurred over one cerebral hemisphere. Examination with the ophthalmoscope may sometimes show that meningitis is present in cases of cerebral rheumatism.

Dr. Forget, however, dissents from the opinion that rheumatic meningitis should be considered as a distinct species. Its symptoms, its perils, and its treatment, he says, are the same as those of simple meningitis. He recommends the free use of opium, which has proved most effectual in meningitis attending typhus, pneumonia, and erysipelas. The hyperæmic condition of the brain which is presumed to coexist is no contra-indication, and is not always present. (Vide *New Sydenham Soc. Year-Book*, 1859, pp. 179, 186.)

Let us now briefly call over the names of the several varieties of cerebral rheumatism. They are: 1. the *apoplectic* and *apoplectiform*; 2. the *paralytic*; 3. the *psychical*, or that which has *mental alienation* for its essential feature; 4. the *convulsive*; 5. the *choreic*; and 6. the *meningitic*, or that which is characterized by the occurrence of meningeal inflammation.

What is the *nature* of the cerebral disorders which are produced by rheumatism? The apoplectic variety is generally due to cerebral embolism; sometimes, however, it is not attended with any lesion of the brain that is discoverable after death. The apoplectiform variety is not characterized by any post-mortem changes. The paralytic variety sometimes results from cerebral embolism, and sometimes no anatomical lesion can be found to account for its occurrence. But the psychical, the convulsive, and the choreic forms of cerebral rheumatism are never attended with any peculiar morbid appearance of the brain or its membranes that has yet been recognized. Of all the varieties of cerebral rheumatism only one is ever characterized by the occurrence of intra-cranial inflammation, namely, the meningitic. In most of the cases where rheumatism occasions cerebral disturbances, no corresponding lesion of the brain or its appendages is found on examination after death. Hence, cerebral rheumatism is, for the most part, a neurosis and not an inflammation. Trousseau says: "I hold to the opinion that the phenomena of cerebral rheumatism are, in general, those of a neurosis much more than of an inflammation or even a congestion having definite anatomical characteristics which can be easily made out." (Vide *Lectures on Clinical Medicine*, vol. i. p. 534, New Sydenham Soc. translation.) Dr. Fuller thinks that delirium, convulsive disorders, and coma, when they occur in consequence of gout or rheumatism, are generally not due to inflammation of the brain or spinal cord, but to a vitiated condition of the blood acting on a nervous system in a state of exalted sensibility. (Vide *New Sydenham Soc. Year-Book*, 1862, p. 155; also *Lancet*, December 27, 1862.)

Another point in the history of articular rheumatism which should not be overlooked is, that the attack is sometimes ushered in with cerebral symptoms. This happened in several instances belonging to the paralytic and choreic varieties of cerebral rheumatism which we have mentioned. It is probable that cerebral rheumatism occurs without any preliminary affection of the joints much oftener than is generally supposed; and sometimes attacks of so-called congestive apoplexy may be due to rheumatic poison in the blood, as well as to that of gout. Generally, however, the disease has existed in the joints for several days, or for even a much longer period, before it affects the brain.

The occurrence of cerebral rheumatism is, in general, not occasioned by metastasis. Trousseau, after making a critical exposition of the subject of metastasis, says: "One may often be misled into believing that rheumatism has left the joints when it attacks the brain, from the severity of the cerebral symptoms masking that of the joint affection. The patient, in his delirium, tosses about wildly, moving in every direction the limbs which he had previously kept motionless on account of the pain in his joints; and, because he is no longer conscious of this pain, those about him believe that his joints are no longer affected. But this is evidently a mistake; the articular rheumatism still persists, for there are still swelling and redness, and exquisite sensibility; but the latter is masked by the delirium, and by the different nervous condition in which the patient is. Under whatever aspect we view the question, therefore, whether in the light of general pathology or in that of the phenomena observed in the course of an attack of cerebral rheumatism, we are justified in regarding the implication of the brain in rheumatism as not due to metastasis."

(Vide *Lectures on Clinical Medicine*, vol. i. pp. 536, 537, New Sydenham Soc. translation.)

We have elsewhere shown that the occurrence of cerebral rheumatism depends on the existence of a special predisposition, which may be either acquired or hereditary, and have already discussed the predisposing causes of this disorder at sufficient length. Lebert finds, from his clinical experience at Breslau, that it occurs in males much more frequently than in females; also that the season of the year and the age of the patient do not seem to exert any notable influence in its production. (Vide *New Sydenham Soc. Year-Book*, 1862, p. 57.)

The predisposing causes of cerebral rheumatism are mentioned above; the exciting cause, however, is the rheumatic poison in the blood. On the latter point Dr. Fuller says: "In all cases in which cerebral disturbance presents itself during the course of acute rheumatism, the altered condition of the blood is its primary or proximate cause; . . . neither delirium nor coma is necessarily accompanied by any internal inflammation, whether of the brain, the heart, or the lungs." (Vide *Fuller on Rheumatism*, etc., p. 293, 3d ed.)

Treatment.—There is no remedy, so far as known, which is a specific for rheumatism. The etiology of cerebral rheumatism affords but few indications to guide its treatment, since its causes are for the most part of such a nature that we cannot remove them, and can never do much to obviate their effects. Sometimes we endeavor to entice the disease away from the brain by the use of derivatives, but generally without any perceptible effect. Thus we are practically restricted to following the symptomatic indications. It is our highest duty to obviate the tendency to death, for there is always reason to hope that, if life can be sufficiently prolonged, the rheumatic poison will become eliminated from the blood, and the integrity of the cerebral functions will be restored. The treatment must, therefore, be adapted to the requirements of each individual case. Sometimes it should be cooling and sedative, at others analeptic and stimulant. If the cerebral symptoms are at all dangerous, and result from an excessive increase of the bodily temperature, it will be highly advisable to promptly reduce the temperature by wrapping the body in wet sheets, by cold affusion, or by cool baths; and these procedures should be repeated as often as the temperature threatens to increase to a great height, as we have already shown. When the temperature is not greatly increased, the application of cold water, frozen compresses, or the ice-bag to the head, will probably suffice. If the pulse is full and strong, veratrum viride can generally be administered with advantage as a sedative to the cardiac action and to the nervous excitement.

To the great value of veratrum viride as an antipyretic, and as a quieter of both nervous and vascular excitement in cases of acute articular rheumatism, the author can bear witness from his own experience. Again, the great value of cold affusions and baths in the treatment of rheumatic hyperpyrexia has lately been shown by a case related to the Clinical Society of London by Dr. Weber, (*Med. Times and Gaz.*, March 23d, 1872,) in which, on the thirteenth day, the temperature suddenly rose to 108.2° F., the pulse to 148, and the respirations to 56, and simultaneously great restlessness, vomiting, delirium, tendency to coma, excessive micturition, and involuntary discharges made their appearance. The patient was kept for thirty minutes in a bath of 71° F., and at the same time affusion of the same temperature was practised. When removed from the bath, the mouth temperature had fallen to 101.8° F., and in ten minutes went down to 98.8°. The patient then went to sleep quietly, perspiring greatly. In the afternoon of the same day the temperature rose again, and the bath and affusion were again resorted to, with the result of reducing the temperature to 101°, and a fall during the next hour of three more degrees. After the second bath the disease took the form of a mild rheumatic fever. (*New Remedies*, July, 1872, p. 34.)

Sulphate of quinine is also a valuable antipyretic remedy when administered in large doses. Trousseau mentions a case of acute cerebral rheumatism, recorded by Dr. Mesnet, in which sulphate of quinine in gradually increasing doses was prescribed, and a remarkable improvement followed. "The choreic movements, the agitation, the hallucinations, the delirious fancies, ceased, but the intellectual confusion continued for another fortnight. After that it disappeared of itself by degrees; health and strength returned, and a complete cure was at last obtained after two months and a half." (*Op. cit.* p. 526.) Dr. Bean mentions a case of cerebral rheumatism, in which he persisted in the administration of quinine, and the patient recovered. (Trousseau.) It was probably an acute or sthenic case, and the quinine did good in consequence of its antipyretic properties. General blood-letting should not be employed. The treatment should, in general, not be too lowering, and mental repose should be maintained. Trousseau states that he has succeeded in curing three patients, who were under his care from the onset of the cerebral rheumatism, by means of musk and opium, but has failed in other instances; and twice he has seen recovery take place without the employment of any active treatment. (*Op. cit.* p. 540.) Opium or morphia is likely to prove very useful in those cases where much pain or restlessness is present, and in the first stage of those cases where meningeal inflammation has occurred. The appearance of hebetude or coma will generally contra-indicate its employment. The bromide of potassium, sodium, or ammonium may sometimes be given with advantage to quiet nervous irritation and procure sleep in the non-inflammatory cases. When in the meningitic variety of cerebral rheumatism effusion of serum has occurred, and, in consequence thereof, hebetude, stupor, coma, dilatation of the pupils, strabismus, etc., have made their appearance, the administration of iodide of potassium in full doses may prove beneficial.

Dr. Chambers relates the case of a letter-carrier, aged 24, who, from the very beginning of an attack of acute articular rheumatism, became violently delirious. He remained so four or five days after admission. The joints were swollen and red during the delirium, and the redness and swelling disappeared simultaneously with the delirium. There was nothing in the man's antecedents to account for the delirium. He had no pericarditis, no endocarditis, no pneumonia. He recovered under a treatment consisting of potass. iodid. in large doses, ammonia and bark, wine and beef-tea every two hours. (Vide *Dr. Bazire's Notes to Trousseau's Lectures on Clinical Medicine*, vol. i. p. 540.) This case was doubtless asthenic in character.

Dr. Fuller recommends the administration of diffusible stimulants and opiates combined in these cases, provided the disturbance in the functions of the nervous centres is ascertained to be functional only, and unconnected with any cardiac, pulmonary, cerebral, or spinal inflammation. He says: "Even local depletion, though sometimes expedient in such cases, for the relief of active local inflammation, should be seldom practised to any great extent. We should rather endeavor to support our patient, [with nourishing food,] whilst aiming at the relief of the more urgent symptoms by means of blisters, mercurials, diuretics, and opium. . . . . One exception only exists to the full though cautious exhibition of opium, ... [namely, when] there is a tendency to the supervention of coma. In such cases opium is not only useless, but it is decidedly prejudicial to the safety of the patient, who requires a more than usual amount of support and stimulus." (Vide *Fuller on Rheumatism*, etc., pp. 314, 315, 3d ed.)

Lemon-juice, taken in the form of lemonade, often assists materially in shortening the attack. The choreic form of cerebral rheumatism may require the administration of conium (succus inspissatus conii) or the extract of Calabar bean; and, all other means for quieting the choreic movements and procuring sleep having failed, it may be necessary to give anæsthetics by inhalation for that purpose. Henoch found arsenic the most serviceable remedy for rheumatic chorea, as already stated.

When cerebral embolism occurs as a consequence of rheumatism it should be treated in the way described in Chapter VI.

Rheumatic apoplexy, when it is not due to cerebral embolism, should be treated on the principles mentioned above. We should strive to obviate the tendency to death in such cases by the use of derivatives, by the application of cold to the head, or even to the whole body when the temperature rises to a great height, in some of the ways mentioned above, and by the administration of diffusible stimulants if the pulse is low, or the heart's contractions weak. The employment of the cold bath and cold affusions will be indispensable in all cases of this sort where the temperature rises to 105° F. and upward, as the cases which we have related abundantly show.

CHAPTER VIII.

ON INFANTILE APOPLEXY, AND INFANTILE CEREBRAL HEMORRHAGE.

They differ widely from the corresponding disorders in adults, and occur much more frequently than is generally supposed.—Infantile apoplexy and infantile cerebral hemorrhage are not synonymous terms.—Two statistical tables given to illustrate the frequency with which infantile apoplexy occurs during each year up to five.—Another table to illustrate the frequency with which apoplexy occurs during each period of five years throughout life.—Physiological anatomy of the infantile brain.—Some reasons given why apoplexy occurs so frequently in young children.—The efficient cause or pathogeny of infantile apoplexy described.—*Of Simple Apoplexy occurring in Infancy and Childhood.*—1. It may result from active hyperæmia of the brain.—2. It may result from passive hyperæmia of the brain.—3. It is often met with in pale-looking, badly-nourished children.—*Case* XLVI. Apoplectic stupor suddenly developed in a weakly and ill-fed child of two years; death; autopsy; well-marked anæmia of the cerebral substance; œdema of the membranes, etc., etc.—*Case* XLVII.—Chronic marasmus occurring in an infant of four months; purpura hemorrhagica; sudden coma and death; autopsy; anæmia of the brain-substance, and œdema of the membranes; blood thin and watery; mesenteric glands enlarged, etc.—4. Apoplectiform phenomena are sometimes produced in children by latent hydrocephalus.—*Case* XLVIII. Apoplectiform symptoms occurring in a boy eight years old; death; autopsy; found cerebral congestion, copious effusion of serum in the ventricles, etc.—5. Infantile pneumonia may, at the outset, be attended with loss of consciousness and other symptoms of apoplexy.—*Case* XLIX. Apoplectiform phenomena accompanying an attack of pneumonia in an infant of eleven months; death from the latter disease; autopsy; found œdema of the meninges and extensive hepatization of the lungs, etc.—6. Apoplectic coma occasionally supervenes in the course of the inflammatory diarrhœa of infants; an illustrative case related.—Again, infantile diarrhœa sometimes induces thrombosis of the sinuses of the dura mater.—*Case.* L. Profuse diarrhœa occurring in a child three months old; stupor, unconsciousness, opisthotonos, and other head-symptoms; death; autopsy; found thrombosis of superior longitudinal and both lateral sinuses; hyperæmia of the meninges, pneumonia, etc.—*Of Cerebral Hemorrhage occurring in Infancy and Childhood.*—Cruveilhier has specially studied the so-called "apoplexy of the newly-born;" his conclusions stated; a brief summary of his cases given; Dr. J. Lewis Smith's observations support Cruveilhier's views; Dr. Smith's cases mentioned; Dr. West's remarks on this subject.—But cerebral hemorrhage may occur at any other period of infancy and childhood. *Case* LI. Bronchitis (acute) followed by head-symptoms; purpura hemorrhagica; death; autopsy; extensive meningeal hemorrhage, etc.; blood thin, watery, and deficient in plasticity. —*Case* LII. Cachexia, convulsions with great depression, purpura hemorrhagica, and speedy death; autopsy; extensive meningeal hemorrhage; anæmia of the brain-substance, etc.—Another case of passive hemorrhage into the arachnoid related. Three other cases of meningeal hemorrhage occurring in infants briefly mentioned.—Occasionally the cerebral substance is the seat of extravasation.—Cases reported by Billard, Serres, Wythes, and Richard Quain referred to.—*Case* LIII. Vomiting, stupor, convulsions, and partial hemiplegia occurring in a girl of eleven years; death; autopsy; found extensive hemorrhage in the cerebral substance and beneath the arachnoid, etc.—*Case* LIV. Scarlatina, anasarca, chronic peritonitis, and pleurisy; death apparently from syncope; autopsy; found thrombosis of the sinuses of the dura mater; four clots of extravasated blood in the brain-substance, etc.—*Case* LV. Diarrhœa and vomiting in a child three weeks old, succeeded by collapse, head-symptoms, cyanosis of the face, and death in three days; autopsy; found thrombosis of the sinuses of the dura mater; extensive hemorrhage into the cerebrum and meninges; anæmia also of pia mater and brain, etc.—*Case* LVI. Typhoid fever occurring in a girl of twelve years: convulsions, purpura hemorrhagica, and death; autopsy; found thrombosis of superior longitudinal sinus; very extensive meningeal hemorrhage; blood extravasated also into the substance of right cerebral hemisphere.—Three other cases in which cerebral hemorrhage was caused by thrombosis of the cerebral sinuses, briefly sketched.—*Ætiology of Infantile Cerebral Hemorrhage.*—It occurs much oftener in weakly children than in robust ones.—Influence exerted by thrombosis of the cerebral sinuses, and by purpura hemorrhagica, in producing it.—Concerning the so-called primary or idiopathic form of cerebral hemorrhage; it occurs but very rarely in young children; Henoch, however, has related three cases.—*Symptoms of Infantile Cerebral Hemorrhage.*—They are very obscure.—Paralysis or hemiplegia but seldom present in such cases.—The reasons given.—Infantile hemiplegia generally due, not to cerebral hemorrhage, but to cerebral tuberculosis.—The symptoms stated which may lead us to suspect the occurrence of cerebral hemorrhage in children.—*Treatment of Infantile Cerebral Hemorrhage.*—When produced by thrombosis of the cerebral sinuses or by purpura hemorrhagica no treatment avails.—How determination of blood to the head or active hyperæmia should be treated; venesection, leeches, purgatives, cold affusion of head.—How the tendency to death may be obviated.

WHEN apoplexy or cerebral hemorrhage occurs during the infantile period of life, but more especially in very young children, its pathogeny, anatomical lesions, symptoms, course, and consequences are so widely different from what they are in the adult period as to require a separate description, and to make it advisable to devote a separate chapter to that purpose.

We stated in Chapter III. that infants perish of apoplexy much oftener than is generally supposed, that sixty-eight children of less than ten years died of it in the city of New-York in 1867, 1868, and 1869, that forty-three, or almost two thirds, of them were less than one year old, and we promised to resume the consideration of this subject in a chapter specially set apart for it. We should, however, remark, before proceeding further, that infantile apoplexy and infantile cerebral hemorrhage are not synonymous terms; and that intra-cranial hemorrhage when it occurs in a young child is much less likely to induce paralysis or hemiplegia and apoplectic coma, than when it occurs in an adult, for reasons of an anatomical character which will be given in another place. We shall, therefore, give an account of apoplexy, properly so-called, as it presents itself in infantile subjects, before we take up the cerebral hemorrhages which are met with in the same class of subjects. We are also strongly impressed with the belief that infantile apoplexy is more important in a practical as well as in a purely scientific point of view than is generally admitted, and that it deserves a more copious description than is usually accorded to it in works on the diseases of children. Dr. West remarks that *simple* apoplexy is by no means rare in childhood, (vide *Lectures on the Diseases of Infancy and Childhood*, p. 57, Am. ed. 1868,) but he does not tell us how frequently it occurs, nor does he relate more than two or three cases which might be termed instances of it, and they are given for another purpose. The following tables, however, will aid us in forming an idea with regard to how frequently it presents itself in children in general, and how often it occurs in each year of infantile life up to the age of five. Cases of *sanguineous* apoplexy (so-called) are doubtless embraced in these tables as well as those of a *simple* character.

Consolidated report of the deaths from Apoplexy occurring among infants of less than five years in the city of New-York, for 1867, 1868, *and* 1869.

	Under one Year.	One.	Two.	Three.	Four.	Total under Five Years.
Males..........	21	8	4	1	1	35
Females........	22	6	0	0	2	30
Total..........	43	14	4	1	3	65

In order to show that the occurrence of infantile apoplexy is not exceptional with us in New-York, the following table is presented :

Registrar-General's report of the deaths from Apoplexy occurring among infants of less than five years in the city of London, for the year 1866, (*Tanner.*)

	Under one Year.	One.	Two.	Three.	Four.	Total under Five Years.
Males..........	44	29	17	12	9	111
Females........	45	13	7	9	7	81
Total..........	89	42	24	21	16	192

These two tables give a grand total of 257 cases, of which 132, or rather more than one half, were less than one year old; 56, or more than one fourth and less than one third, were between one and two; 28, or about one ninth, were between two and three; 22, or rather more than one twelfth, were between three and four, and 19 were between four and five; the yearly ratio rapidly diminishing after the first year.

Again, a careful analysis of the reports of the Metropolitan Board of Health of New-York on the subject of apoplexy, for the years 1867, 1868, and 1869, shows that of the whole number who were destroyed by it during this period of three years—

65	were under	5 years.					97	between	45	and	50	years.
3	between	5	and	10	years.		80	"	50	"	55	"
5	"	10	"	15	"		106	"	55	"	60	"
10	"	15	"	20	"		115	"	60	"	65	"
20	"	20	"	25	"		94	"	65	"	70	"
40	"	25	"	30	"		85	"	70	"	75	"
59	"	30	"	35	"		47	"	75	"	80	"
87	"	35	"	40	"		21	"	80	"	85	"
80	"	40	"	45	"		11	"	85	"	90	"

These reports also show that scarcely any single year in the life of man is more prolific in deaths from apoplexy than the first one; that after the first year has been passed, the mortality from this disease rapidly diminishes, until the age of puberty; that after this epoch it steadily increases until the age of sixty or thereabouts, and that after this time it slowly declines.

The physiological anatomy of the infantile brain and its coverings explains, in part at least, why apoplexy occurs so much more frequently during the first year or two of infantile life than in the later portions of the same period. At birth the brain, compared to that of the adult, is very large, very soft, somewhat undeveloped in structure, and rather watery in appearance. The convolutions are in a rudimentary state, and the furrows but faintly marked. The membranes and the substance of the brain itself are more vascular than they are at the adult period. The bones which constitute the cranial vault or dome are joined together, not by cartilage and osseous tissue, as they are after the sutures are formed, but by a membranous substance which readily allows them to overlap each other. There are also two large gaps in the skulls of the newly-born, near the summit, called fontanelles, which are bridged over by a very strong and flexible membrane. These fontanelles and the membranous union between the cranial bones themselves permit great expansions and contractions in the volume of the cerebral circulation to suddenly occur without destroying the little patient at once, as they would be apt to do if the cranial walls were fixed and unyielding, as they are in the adult period of life. At birth, the brain weighs about 10 oz., (*Tanner*,) and constitutes from $\frac{1}{10}$ to $\frac{1}{15}$ part of the weight of the whole body; while in adults the average weight of this organ is 49½ oz., or a little more than 3 lbs. avoirdupois, and constitutes only from $\frac{1}{40}$ to $\frac{1}{50}$ part of that of the whole body.

Furthermore, the nutritive activity in the infantile brain is very great, and it grows with wonderful rapidity. "There is no organ in the body, with the exception of the pregnant womb, which undergoes such rapid development as the brain in early childhood. It doubles its weight in the first two years of life, and reaches nearly, if not quite, its maximum by the end of the seventh year." (Vide *West's Lectures on the Diseases of Infancy and Childhood*, pp. 37, 38, Am. ed. 1868.) Another writer of eminence says the weight of the brain increases rapidly up to the seventh year, more slowly to the period between 16 and 20, and still more slowly to that between 30 and 40, when it reaches its maximum. Beyond this period, as age advances and the mental faculties decline, the brain diminishes slowly in weight, about an ounce for each subsequent decennial period. These results apply alike to both sexes. (Vide *Gray's Anatomy*, p. 510, 2d Am. ed.) The remarkable activity in the processes of nutrition and growth which characterizes the infantile brain renders the organ not only very vascular, but also liable to have morbid action excited in it by causes which would produce little or no effect upon the adult brain. (*West.*)

Now, why does apoplexy occur so much oftener in very young children than it does in those that are four or five years old? The answers to this question are several in number.

1. The substance of the brain is softer and affords less support to the cerebral blood-vessels, and therefore they are more liable to be ruptured by intra-vascular pressure, at the former period than at the latter.

2. The sutures and fontanelles are much more flexible and disposed to yield in the young child than they are in one that is several years old. When cerebral hyperæmia presents itself in the young child, this circumstance favors the occurrence of engorgement and stagnation of blood in the cerebral vessels, together with arrest in the supply of freshly oxygenated blood to the ganglion-cells and nerve-fibres of the brain. When, on the other hand, cerebral anæmia occurs in the young child in consequence of hemorrhage, or of diminished heart-power, or of starvation, or of exhaustion from abdominal disease, or from any other cause, the open state of the sutures and the fontanelles causes the anæmic condition of the brain-substance to be greatly aggravated by atmospheric pressure acting upon or through the flexible walls of the cranium in such a way as to compress the cerebral capillaries and prevent the circulating blood from entering them. Thus, the condition of the sutures and fontanelles which at one time ex-

poses the infant's brain to be overfilled with blood, at another may cause it to become more completely exsanguinated or anæmic than is possible in an older subject. This fact, which should never be forgotten by those who treat the diseases of infants, affords one reason why excessive depletion induces a far more dangerous train of symptoms in young children than it does in grown persons. (*West.*)

3. It is probable that œdema of the brain-substance and of the meninges occurs more readily in young infants than it does in those who are several years old.

The efficient cause of apoplexy is the same whether it occurs in young children or in grown people, namely, anæmia of the nerve-fibres and ganglion-cells of the brain. This point in the pathology of apoplexy was pretty fully elucidated in Chapter II. We shall make only a brief allusion to this subject in this place. When apoplectic coma occurs in a young child it is because the functions of its brain are suddenly suspended, and these functions are suspended because the nerve-fibres and ganglion-cells do not receive freshly-oxygenated blood enough to keep them in a state of activity. When cerebral congestion or hyperæmia produces apoplexy in an infant it is because the blood stagnates in the swollen vessels of the brain, and thus precludes the nerve-fibres and ganglion-cells from receiving that supply of freshly-oxygenated blood which is indispensable to the performance of their functions. The fresh arterial blood cannot get into the brain, because the effete venous blood does not go out of it, and thus, in such cases, the brain-substance is really in a state of anæmia, although the large arteries and veins are at the same time filled to distention. When cerebral œdema gives rise to apoplectic symptoms in children it is because the serum is effused in the perivascular spaces of the brain, or in the ventricles, or in the membranes, or simultaneously in all of these situations, in such a way as to compress the cerebral capillaries and put a stop to their conveyance of blood to the nerve-fibres and ganglion-cells. In such cases the brain-substance generally presents an exsanguinated or anæmic and moist or shining appearance. Again, when apoplectic coma occurs in an infant that is much debilitated from loss of blood or from excessive evacuations, whose pulse is weak and circulation feeble, the tendency to the occurrence of cerebral anæmia is much increased by atmospheric pressure acting upon or through the membranous sutures and fontanelles and compressing the capillaries of the cerebral substance so as to exsanguinate them, as we have already shown on one of the preceding pages. Finally, when coma is produced by cerebral hemorrhage in children, it is not because the extravasated blood exerts pressure upon the cerebral substance itself, but because it compresses the cerebral capillaries and thus induces anæmia of the nerve-fibres and ganglion-cells. This subject has been thoroughly discussed in Chapter II., and to it the reader is again respectfully referred.

1. Of simple Apoplexy occurring in Infancy and Childhood.—Coma, when suddenly produced in young children by some disturbance of the brain that is not hemorrhagic, nor inflammatory, nor traumatic in its origin, is the characteristic feature of simple infantile apoplexy. Dr. West remarks, as we have already stated, that this affection is by no means rare. There are several distinct varieties of it, and some of them differ from each other very much in a clinical point of view.

(1.) Simple apoplexy is sometimes met with in children as a consequence of cerebral congestion or hyperæmia of an active character. Dr. West says: "Convulsions and apoplectic symptoms sometimes come on suddenly in a child previously, to all appearance, in perfect health, and may even terminate in death in less than twenty-four hours. The brain is found loaded with blood, but all the other organs of the body are quite healthy. Some years ago, I was requested to be present at the examination of the body of a boy not quite two years old, who had been in perfect health until the day before his death, which took place in such circumstances as I have just mentioned. The congested state of the cerebral vessels gave but little satisfactory information; but the same evening, the brother of the child was taken ill with vomiting, intense fever, and sore throat. In a few hours a red rash appeared; the case was one of scarlet fever, and ran its course with considerable severity, though, happily, to a favorable termination. It is probable that the poison of the fever had affected the blood of both children, and that the consequent disturbance of the cerebral circulation was so violent as at once to destroy the life of the younger, while the elder brother survived the shock, and in him the disease soon presented its usual features. The history of most epidemics of scarlatina would afford other instances of a similar nature." (Vide *Lectures on the Diseases of Infancy and Childhood*, pp. 46, 47, Am. ed. 1868.)

The same author relates another important case belonging to the same category: "I was called one day to see a little girl two years old, who, until the day before, had never had an hour's illness. She had eaten a hearty dinner, and, though she vomited soon afterward, did not seem otherwise indisposed, and slept well in the night. Immediately on waking in the morning, however, she had a fit, during which she was insensible, squinted, threw her limbs about, and occasionally screamed aloud. She continued very ill through the whole day; was hot and feverish during the night, having occasional attacks of convulsions, in which she stretched out her legs, threw back her head, now and then uttered a word or two, and then relapsed into a state of insensibility. This was her condition at half-past 10 A.M., about twenty-four hours after the occurrence of the first fit. I bled her to three ounces, and would have drawn more blood if it had continued to flow; and then put eight leeches on her head, employed cold affusion, and gave active cathartics through the day, but without much benefit; and at midnight she was still insensible, rolling uneasily from side to side, boring with her head in the pillow, squinting, and making automatic movements with her mouth and tongue. I now put eight leeches more on her head, which bled profusely, and the bleeding was followed by great diminution in the convulsive movements. About 4 A.M. of the next day, the child fell asleep, and dozed for a few hours. She awoke sensible and continued so. On my visit in the morning, I found her quiet and sensible, without any sign of convulsion; her face was very pale; her head, before so hot, was now quite cool; her pulse had sunk in frequency, and lost its fulness. An eruption of a papular character had appeared on the hands, arms, inside of the thighs, and slightly on the face. This eruption was the small-pox, and the disease ran its course with no unfavorable symptoms." (Vide *Lectures on the Diseases of Infancy and Childhood*, p. 47, Am. ed. 1868.) In another place, Dr. West mentions this case again, and says she would most likely have died of apoplexy if she had not been bled very freely; and he also thinks it probable that the depletion might have been carried still further with advantage to the patient. (*Op. cit.* p. 50.)

But active congestion of the brain of a very dangerous character not unfrequently occurs in children who are not affected with scarlet fever or small-pox or any other exanthematous disease; and if in these cases as well as in those quoted above, says Dr. West, "relief be not afforded by appropriate treatment, death is seldom delayed beyond forty-eight hours from the first fit, though no graver lesion may be discovered afterward than a gorged state of the vessels of the brain and its membranes, and perhaps a little clear fluid in the ventricles and beneath the arachnoid." (Vide *op. cit.* p. 49.) It is probable, however, that when fluxionary hyperæmia or active congestion of the brain produces apoplectic coma, œdema of the cerebral substance, or effusion of serum into the perivascular spaces of the brain itself, is also present in such degree as to compress the cerebral capillaries, and paralyze the nerve-fibres and ganglion-cells by depriving them of their blood-supply. This subject has already been pretty fully discussed in Chapter II., and the reader is again respectfully referred to that chapter for further information concerning it.

(2.) Simple apoplexy occasionally occurs in children as a result of cerebral hyperæmia of a passive character. Dr. West says: "In children, who have suffered long and severely from whooping-cough, you often notice a general lividity of the face and lips, a puffed and anxious countenance, and the child makes grievous complaints about its head, while the skin is moist and cool, and the pulse soft though frequent. Many of these symptoms indicate an over-loaded state of the cerebral veins; and if a paroxysm of coughing occur, and the circulation be thus further disturbed, the child may die in a fit, or may sink, after some convulsive seizure, into a state of coma, which sooner or later proves fatal. In such a case you will find the vessels of the brain and its membranes universally gorged with black blood, the choroid plexus of a deep purple color, and more bloody points than natural will present themselves on a section of the brain being made. Both the symptoms during life and the appearance after death are only a rather exaggerated illustration of what occurs in all cases of passive congestion of the brain." (Vide *Lectures on the Diseases of Infancy and Childhood*, pp. 54, 55, Am. ed. 1868.) As mentioned above, death sometimes occurs in these cases, with the symptoms of apoplexy. In such cases the apoplectic phenomena are due to the fact that the nerve-fibres and ganglion-cells do not get a sufficient supply of freshly-oxygenated blood, because the cerebral capillaries are, to a great extent, gorged with stagnant venous blood.

(3.) Apoplexy is often met with in pale-looking, badly-nourished children, and, on examining the fatal cases after death, the brain is found to present an exsanguinated or anæmic appearance, with more or less œdema of its membranes. The next two cases, the notes of which were taken by the author at the time of their occurrence, belong to this category.

Case XLVI.

Apoplectic stupor suddenly developed in a young child; death; autopsy; extensive cerebral anæmia; œdema of the membranes; minute extravasations of blood on convex surface of both cerebral hemispheres.

A male infant, aged two years, was admitted to the Emigrants' State Hospital, Dr. Wendell's wards, about December 7th, having the symptoms of a slight gastric fever; besides, it was pale, thin, weak, and suffering apparently from the want of suitable food; nothing but a good diet was prescribed. It seemed to be doing well till the morning of December 14th. While sitting up in bed, eating breakfast, on that morning, it suddenly fell over, or fainted, as it were, and at once became unconscious. Dr. Wendell saw it soon afterward, and found it lying in deep stupor; it could be roused somewhat by making very strong impressions; face pale; pupils widely dilated, the right rather more than the left; respirations very frequent, much labored, and attended with rattles in the large air-passages, which could be distinctly heard when standing by the bedside. It did not recover consciousness, but died comatose the same evening. It did not have any convulsions.

Autopsy, by the author, eighteen hours after death.—Cadaver emaciated, and of a dingy, yellowish hue; pupils as described above. The brain and its membranes contained less than the normal quantity of blood, that is, they were exsanguinated. On the convex surface of both cerebral hemispheres a number of dark-brown spots, having the size of a split pea, were found. They resembled ecchymoses in appearance, and consisted of minute extravasations of blood in the meshes of the pia mater. They were not found in any other part. There was a moderate amount of serous infiltration or œdema in the subarachnoid connective tissue along the track of the superficial blood-vessels. The anterior cerebral lobes appeared to be somewhat softer than natural. There was much more than the usual quantity of serum about the cerebellum, and the substance of this part of the brain was generally softer than natural. The throat and larynx were not obstructed. The lungs and heart were not diseased. The spleen was almost as large as that of an adult. The organs generally contained much less than the normal quantity of blood. There was commencing gangrena oris, or noma.

Comments.—The hæmic spots which were found on the convex surface of the cerebrum in this case were the results of capillary hemorrhage in the pia mater; but the quantity of blood extravasated was not enough to produce any considerable disturbance of the cerebral functions nor any important symptoms. They were probably formed while the patient was in the article of death; and it is barely possible that they were the result of a thrombosis of the superior longitudinal sinus, which may have been overlooked at the autopsy. There was well-marked anæmia of the brain-substance, and an œdematous condition of the meninges, which reminded one not a little of the changes which are sometimes observed in cases of so-called serous apoplexy occurring in adults. Paralysis of the brain resulting from anæmia of the brain-substance was the cause of death.

Case XLVII.

Chronic marasmus occurring in an infant; purpura hemorrhagica; sudden coma and death; autopsy; anæmia of the brain and œdema of its membranes.

Annie McGuire, aged four months, had been wasting away for some time, in consequence of mesenteric disease. She died suddenly and rather unexpectedly of coma, December 15th, in Ward No. 4, of the Emigrants' State Hospital; did not have convulsions.

Autopsy, by the author, twenty-four hours after death.—Cadaver much emaciated and dirty yellowish in color; scalp thickly covered with the scabs of crusta lactea; legs and arms thickly dotted with small purpuric spots; thumbs and great toes rigidly flexed. The brain and its membranes presented an exsanguinated appearance. There was considerable serum effused beneath the arachnoid membrane, especially along the track of the optic nerves, about the medulla oblongata, and at the base of the cerebellum. The lateral ventricles contained a small quantity of serum. The right hemisphere of the cerebellum appeared to be somewhat softened. The lungs contained less blood than usual. The blood in all parts of the body was thinner and more watery than natural. The mesenteric glands were enlarged.

Comments.—Although the patient had many spots of purpura in the skin, there were no spots of a similar character in the pia mater. The blood was thin and watery. It is also worthy of remark, that in both this and the preceding case there was an utter absence of convulsive movements. Now, young children are extremely liable to get convulsions on account of the remarkable excitability of their motor nervous apparatus. For example, in early life convulsions may be induced by constipation, by worms, by renal calculus, by fright, or by pressure from a growing tooth upon the swollen gum. Dr. West also says that convulsions generally occur at a very early period in cases of infantile apoplexy and infantile cerebral congestion, when it is intense or severe. Our observations, however, show that they are not always present in cases of infantile apoplexy.

(4.) Apoplectiform phenomena are sometimes produced in children by latent hydrocephalus. The next case will serve to show the correctness of this opinion.

CASE XLVIII.

Apoplectiform phenomena occurring in a young boy; death; autopsy; cerebral congestion; serous effusion in the ventricles, etc.

John Carroll, aged eight years, admitted to the Emigrants' State Hospital, Dr. Wendell's wards, November 7th. Nothing of his previous history was known. At time of admission he was comatose, and lay in that state till November 12th, when he died; did not have any convulsions; did not notice surrounding objects; eyes half-closed, and everted; pupils dilated; stupor very profound, as he could not be roused from it even for a single moment. Dr. Wendell also informed us that he did not discover that any part was paralyzed except the iris, as the pupils would not contract even when a lighted candle was brought close to the eyes, and the patient seemed entirely sightless.

Autopsy, by the author, forty-eight hours after death.—Brain and its membranes much congested. But little fluid in cavity of arachnoid. Several ounces of pale serum in lateral ventricles. The cerebral substance surrounding these ventricles was softened and of a cream-like consistence to considerable depth. Other organs healthy.

Comments.—The autopsy showed that hydrocephalus was the disease which produced the loss of consciousness or stupor and coma in this case, and in the end destroyed the patient's life. The copiousness of the serous effusion which was found in the lateral ventricles, and the softened state of the cerebral substance surrounding them, were highly characteristic of this disease, and left no room for doubt in regard to the diagnosis. Dr. Gölis, of Vienna, proposed the name of "*water-stroke*" for such cases as the above. (Vide *West's Lectures on the Diseases of Infancy and Childhood*, p. 92, Am. ed.)

(5.) When pneumonia occurs in childhood, it is not unfrequently attended, at the outset, with loss of consciousness, and other symptoms of apoplexy. The history of the following case affords an interesting example.

CASE XLIX.

Apoplectiform phenomena accompanying an attack of pneumonia; death from the latter disease; autopsy; meningeal œdema; extensive pulmonary hepatization, etc.

Michael Mullens, aged eleven months. About two weeks ago he had a severe attack of pneumonia, which was attended with the following head-symptoms, namely, he lay utterly insensible, his pupils were contracted, and his head seemed hotter to the touch than the rest of his body. Two days afterward he recovered sensibility to external impressions, which he preserved till day of death. But the pneumonia continued, and he died of it November 24th. He did not have convulsions at any time, so far as we could ascertain.

Autopsy, by the author, thirty-six hours after death.—A placid smile rested on the countenance. Pupils slightly dilated.

Head.—On removing the skull-cap, about an ounce of serous fluid escaped. There was more serum than natural in the cavity of the arachnoid and in the subarachnoid connective tissue, (œdema of the meninges.) The arachnoid presented an opaque pearly appearance, especially at base of brain; also to considerable extent on the convex surface of the cerebral hemispheres, due probably to imbibition of serum. The lateral ventricles contained a small quantity of clear liquid, and the plexus choroideus was infiltrated with serum.

Thorax.—Lower lobe of left lung completely solidified with red hepatization, passing into the gray stage thereof. Two thirds of both the lower and middle lobes of the right lung were in a state of red hepatization. The bronchia were inflamed. Other organs healthy.

(6.) Apoplectic coma occasionally supervenes in the course of the inflammatory diarrhœa of infants, and leads to disastrous consequences. Several years ago, I attended a little boy, about two and a half years old, for dysenteric diarrhœa, who, with his parents, lived in the Bowery. The disease appeared to readily yield to the treatment employed, which consisted mainly in a carefully-regulated diet, confinement to bed, poulticing the bowels, and administering calomel and Dover's powder in small doses, alternated occasionally with laxatives. At the end of about the third day he was free from fever, his pulse and skin had become natural, his countenance bright, his stools much less frequent and more natural in appearance, and I expected he would make a speedy recovery. The next morning, however, I found him lying in a state of profound coma, with dilated pupils and a very pale countenance. I learned that the diarrhœa had given no trouble through the night, and that he was discovered to be insensible on trying to waken him in the morning. He sank rapidly, and died comatose in the evening. No autopsy. This child was of the lymphatic temperament, and pale-looking, when attacked with the dysenteric diarrhœa.

Dr. West, after speaking of bronchitis as an intercurrent malady which greatly increases the danger arising from the inflammatory diarrhœa of childhood, says: "Life is sometimes cut short by other causes in the course of infantile diarrhœa.[1] The disturbance of the nervous system that attends the attack issues now and then in convulsions, and these convulsions end in a state of stupor which terminates in death—an occurrence fortunately rare, but of which instances may be observed during those hot seasons of the year when bowel-complaints are usually epidemic. Less rare than a fatal termination of this kind is the infant's death under symptoms of a gradually deepening coma, which may have supervened on the suppression of the diarrhœa, or on its great mitigation." (Vide *Lectures on the Diseases of Infancy and Childhood*, p. 516, Am. ed. 1868.)

The next case possesses very great interest, for in it a profuse diarrhœa was followed by insensibility, tetanic spasms, and thrombosis of the sinuses of the dura mater.

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[1] The susceptibility of the brain to morbid impressions is much greater in young children than in grown people. This remark is especially applicable to impressions which are transmitted from the bowels to the brain by reflex nervous action, and *vice versâ*. Dr. Tanner very justly observes: "Between the stomach or alimentary canal and the brain or nervous system there is so intimate a relation that severe functional disturbance (a prelude often to organic disease) of the former is a not unfrequent result of derangement of the latter, through reflex action."

"Again, from causes already mentioned, the circulation through the brain may be easily deranged, and hence we get congestion, inflammation, spurious hydrocephalus, and convulsions. Convulsive disorders arising from functional disturbance of the brain or spinal marrow are often the result of irritation transmitted from the digestive organs, and will only cease on their removal. We have also known fatal convulsions or dangerous cerebral irritation excited by sudden alarm." (Vide *Tanner on the Diseases of Infancy and Childhood*, p. 88.)

Case L.

Profuse diarrhœa ; stupor, unconsciousness, opisthotonos, with other head-symptoms ; death ; autopsy ; thrombosis of superior longitudinal and both lateral sinuses ; hyperæmia of the meninges ; pneumonia, etc.

A well-fed boy, three months old, was seized with profuse diarrhœa. The greater fontanelle flat and pulsating strongly. Temporal and frontal vein strongly developed. Jugular vein on each side much and equally distended. The child lies quietly, with an unconscious stare and occasional strabismus; both pupils equally dilated. Complete unconsciousness with opisthotonos and muscular rigidity followed. The fontanelle sinks in, and the cranial bones overlap each other. Convergent strabismus; somnolence. First, the left jugular vein became more distended; then the right one became enormously so, while the left appeared almost empty; slight paralysis of left side of face; left pupil more dilated. After some transient improvement the child again became worse, with indications of consolidation of the posterior part of the right lung. Death occurred on the eleventh day.

Autopsy.—Extravasation into the subcutaneous cellular tissue at the back part of the head; the posterior half of the cranial bones very hyperæmic. In the superior longitudinal sinus anteriorly, fluid blood and recent coagula; an inch and a half from its posterior extremity commences a knobby, discolored, but not yet softened thrombus; it extends into both lateral sinuses, and is so large that they, but especially the left one, appear externally like thick, roundish, hard cords. These thrombi reach to within half an inch of the jugular foramina, and end in smooth points. The left is partially adherent, and completely fills the sinus; the right, not. Hyperæmia of the pia mater and gray substance of the brain. Patches of pneumonia in lower lobe of both lungs. (*Dr. Gerhardt's case.* Vide *Dusch on Thrombosis of the Cerebral Sinuses*, p. 112, New Sydenham Soc. translation.)

Comments.—Thrombosis of the sinuses of the dura mater, that is, obstruction of these sinuses with coagulated blood, in all probability, plays a much more important part in the cachetic diseases of young children than has generally been supposed. Its occurrence must obviously lead to great disturbance of the cerebral functions. Some half-dozen cases of this affection will be related in the sequel.

2. Of Cerebral Hemorrhage occurring in Infancy and Childhood.—Under this head we have, first of all, to consider the so-called "apoplexy of the newly-born." This disorder is an accident which, for the most part, has its origin in the process of child-birth. It usually con-

sists in the extravasation of blood upon the surface or at the base of the brain, or, in other words, of meningeal hemorrhage. It occurs very frequently; much more frequently, indeed, than cerebral hemorrhage at any other period of infantile life. It is, therefore, the form of cerebral hemorrhage which presents itself most often in infancy and childhood. In it the effused blood is generally found to be liquid and very dark in color.

Cruveilhier has made this accident the subject of special study. From the investigations which he made at the Maternité, touching the cause of death in still-born cases, he thinks, (1,) that cerebral hemorrhage destroys fully one third of the children that succumb during labor, after having been full of life at the commencement of it. He also found that cerebral hemorrhage was present in almost all the cases of still-birth where death is usually referred to asphyxia or to congenital debility.

(2.) He considers that the "apoplexy" of new-born infants is characterized by an extravasation of liquid blood into the cavity of the arachnoid.

(3.) He says that apoplectic infants are not always born dead; in a considerable number of them respiration is more or less completely established, either spontaneously or by the aid of art. Many live twenty-four or forty-eight hours, or even three or four days, in a state of weakness and torpor which is usually ascribed to debility.

(4.) He thinks it impossible to determine the cause of the extravasation in a large proportion of the cases. Sometimes it is due to protraction of the labor, sometimes to constriction of the neck by the umbilical cord, sometimes to compression of the cord itself, as happens, for example, in cases where the cord presents.

(5.) He regards all cases of cerebral hemorrhage occurring in new-born infants, as cases of extravasation resulting from mechanical causes, which it will be possible to prevent, in the majority of instances, by an opportune termination of the labor by artificial means.

Cruveilhier also presents a brief summary of seven cases of this accident which had come under his own observation. They are as follows:

(1.) Infant born dead; presentation vertex in the first position; labor natural, and lasted only sixteen hours. At the autopsy a large quantity of black blood was found surrounding the cerebrum and cerebellum; and some more liquid blood of a less dark color was found in the spinal canal. The only appreciable cause of the hemorrhage was compression of the umbilical cord.

(2.) Infant born dead; presented by the vertex. Suspension of the pains on the escape of the head. Tardy delivery of the trunk. Considerable extravasation of blood in the ventricles of the brain, and their walls are lacerated. Extravasated blood beneath the arachnoid investing the medulla oblongata. Liquid blood in the cavity of the spinal arachnoid.

(3.) Infant born dead after a very long labor, and repeated attempts at version.

(4.) Infant born dead; delivered by the feet.

(5.) Infant died an hour after birth.

(6.) Infant still-born in consequence of presentation of the cord.

(7.) Hydrocephalic fœtus born dead. Labor natural. Cerebral hemorrhage. (Vide *Cruveilhier's Anatomie Pathologique du Corps Humain*, liv. xv. p. 3.)

Cruveilhier's views concerning the apoplexy, so-called, of the newly born, are corroborated by the experience of other observers. For example, Dr. J. Lewis Smith exhibited a brain belonging to this category to the New-York Pathological Society, February 9th, 1870. It was obtained from an infant which had survived its birth for twenty-eight hours, and died of so-called asphyxia. The mother was a primipara. The face presented. The labor lasted thirty-six hours. The delivery was effected without instruments. It is not known whether ergot was administered or not. The infant was apparently still-born, but, after considerable effort, respiration was imperfectly established, and it lived twenty-eight hours, as mentioned above. At the autopsy, blood was found extravasated over the entire surface of the brain, and into its ventricles. The brain was also intensely congested. The lungs were in a state of almost complete atelectasis, the anterior portion of the right lung only being inflatable. Dr. Smith remarked that this was the third case in which he had made a post-mortem examination under like circumstances, and found that profuse meningeal hemorrhage had occurred. In only one of these three cases had instruments (forceps) been used. He believed that, in very many instances, the death of new-born infants was due to this effusion of blood upon the brain, when, in the absence of such effusion, respiration would have been established, and the little one would probably have lived. (Vide *The (N. Y.) Medical Record*, vol. v. pp. 20, 21.)

Dr. West also says: "All periods of childhood are not equally exposed to this accident, [cerebral hemorrhage;] but it is oftenest met with immediately after birth, and no circumstances can be imagined more favorable to its occurrence than those which then concur to produce it. The head of the infant has been subjected to severe and long-continued pressure during its progress through the mother's pelvis; immediately on its birth, the course of the circulation is altogether changed, and, should any difficulty occur in the establishment of the new function of respiration, a long time will elapse before the blood flows freely through its unaccustomed channels. No one will wonder that death should frequently take place during this transition to a new kind of existence. The tumid scalp and livid face of many a still-born child point to one of its most important causes, since they are but the measure of that extreme congestion of the vessels within the skull that has at length ended in a fatal effusion of blood upon the surface, or at the base of the brain.

"There would be reason to fear that this occurrence had taken place, if an infant, when born, were to present great lividity of the surface, and especially of the face, and if the heart were to beat feebly and at long intervals, although the pulsations of the cord were slow and faint, or had altogether ceased. In these circumstances, death sometimes takes place without any effort at respiration being made, the beatings of the heart growing feebler and fewer, till they entirely cease; but at other times the child breathes irregularly, imperfectly, and at long intervals. The hands are generally clenched, and spasmodic twitchings are of frequent occurrence about the face, or these twitchings are more general and severe, and amount almost to an attack of convulsions. The symptoms, however, are by no means uniform, and probably are in some degree modified by variations in the seat as well as in the quantity of the effusion ; for it sometimes happens, even in cases where a very large quantity of blood has been poured out into the arachnoid cavity, that the breathing is little or not at all disturbed, and that, after living for a few hours in a state of weakness and torpor, with chilliness of the whole surface, the child dies without any signs of convulsions." (Vide *Lectures on the Diseases of Infancy and Childhood*, p. 58, Am. ed. 1868.)

But cerebral hemorrhage may occur in new-born infants from the operation of causes which have no relation whatever to the act of parturition. For example, we have already related a case in which copious extravasation of blood into the membranes of the brain (see Case XXX.) occurred in a child two days old. This hemorrhage was probably caused in part by injury in the shape of concussion or contusion of the brain, and in part by convulsive movements of an eclampsic character. This child had appeared to be "pretty smart" on the day after its birth, but on the succeeding morning it was seized with convulsions, etc., and died in the afternoon in a fit. The marks of three distinct bruises were found on the scalp. Some, at least, and perhaps all of them, had been inflicted some little time after birth.

Again, the occurrence of cerebral hemorrhage in childhood is not restricted to the time of birth. It may present itself at any other period, although it occurs much oftener in the newly-born than in those who survive that epoch. Furthermore, the seat of the extravasation, in a large majority of instances, continues to be the arachnoid cavity and the subarachnoid connective tissue. Thus, meningeal hemorrhage is the form of cerebral sanguineous effusion, which is most common in every period of childhood. In adult life, however, just the reverse obtains, since extravasation into the substance of the brain is the form of cerebral hemorrhage which then most frequently occurs. The following case is an instance of profuse meningeal hemorrhage that occurred in a boy of eight years.

CASE LI.

Bronchitis acuta, followed by head-symptoms; purpura hemorrhagica; death ; autopsy ; extensive meningeal hemorrhage, etc.

An unhealthy-looking boy, about eight years old, of whose previous history nothing was known, an inmate of the Nursery on Ward's Island, where he was seen by Dr. Wendell, Nov. 7th, and immediately sent to the Hospital. He had the signs of acute thoracic inflammation, such as flushed face, cough, dyspnœa, dilatation of nostrils at each inspiration, pain in chest, pungent heat of skin, frequent and rather incompressible pulse, etc. He was put on potass. nitrat. et ipecac. in small doses frequently repeated, and had a poultice applied to his chest. In the evening he was no better; same treatment continued.

Nov. 8. Finding no abatement of the symptoms, a vein in his arm was opened and a few ounces of blood taken; same treatment continued. In the evening he was decidedly better. By the 10th, nothing remained but a slight bronchitis. At my first visit (said Dr. Wendell) he looked rather drowsy, but showed no other symptoms of brain-disease.

Nov. 12. He looks quite drowsy; sleeps with his eyes half open; pupils slightly dilated; notices nothing; bawls out in his sleep; respiration natural; pulse frequent and small in volume. He cried and made resistance on attempting to move him. These symptoms became intensified, so that he made obstinate resistance when touched or moved, would not put out his tongue, and was bawling out in his sleep "every half-hour," as the nurse said. The alvine discharges were natural, as also the urine. A blister was applied to the back of his neck, and calomel prescribed.

Nov. 14. Frothy blood filled his mouth, so that the tongue was not seen; skin cool; pulse diminished in force; pupils, as before, slightly dilated; breathing slow and labored. In the evening some purpuric spots appeared, and at midnight he died comatose. He was not restless at any time, nor was there from first to last any sign of convulsive action.

Autopsy, by the author, fifteen hours after death.—Body emaciated, and of a yellowish color somewhat tinged with green. A few purpuric spots on legs and left hip, of considerable size, and without a well-defined margin; when cut into, blood was found effused in skin itself and in connective tissue beneath to considerable depth. Right pupil dilated a little more than the left.

Head.—On convex surface of middle lobe of right cerebral hemisphere a flat-shaped recent clot of blood was discovered, which when gathered into a mass was as large as a filbert. At the corresponding place on the left hemisphere there was another recent clot of much smaller size; and on the posterior surface of the posterior lobe of the right hemisphere there was still another small flat-shaped recent clot. These coagula were all found lying in the cavity of the arachnoid, that is, on its free surface. A little sero-sanguineous effusion at base of brain. Brain-substance and membranes paler than usual; blood contained in their vessels thin and watery. No other morbid appearance in the cranium.

Thorax.—Lungs emphysematous and contain rather more blood than usual, but it is thin and watery; brown spots as large as peas, produced by extravasation of blood, are found scattered through the substance of both lungs on cutting into them; bloody fluid mixed with air (or frothy) is given out under pressure. Bronchiæ inflamed. Each pleural cavity contains a small quantity of bloody serum. Pericardium also contains a small quantity of bloody serum. Substance of heart paler than natural; a few hemorrhagic spots in walls and fleshy columns of left ventricle. Heart-clots very small. Blood throughout body thinner and paler than natural.

Abdomen.—Liver paler than usual, although it contains fully the normal quantity of blood. Gall-bladder filled with green bile. Spleen enlarged, softened, and full of blood; internally it presents a dark-brown, rotten appearance. Kidneys paler than natural; right contains more than the normal quantity of blood, which is thin and pale; otherwise they are healthy. Stomach exhibits a few punctiform hemorrhagic spots scattered over fundus and along greater curvature; pyloric portion corrugated. Œsophagus intensely inflamed to the extent of two or three inches above the cardiac orifice of the stomach. Large intestine blue-black in color from extravasation of blood; it is not gangrenous. At or about the termination of the upper third of the ileum there is an intussusception; invagination from above downward to the extent of one and a half inches; parts implicated not inflamed. Peyer's patches unusually distinct. Mucous membrane of stomach and intestines softened throughout. Pancreas enlarged. Other organs normal. For the clinical history of this very interesting case we are indebted to Dr. Wendell, whose patient he was.

Comments.—In this case the blood was thin, watery, and deficient in plasticity. It is probable that the sanguineous effusion upon the brain and the spots of purpura hemorrhagica in the skin were mainly due to the morbid state of the blood itself; or, in other words, the condition of this patient's blood was such that it strongly favored the occurrence of capillary hemorrhage in all parts of the body. Extravasations of this character were found in many different tissues and organs, as, for example, the membranes of the brain, the skin and subcutaneous connective tissue, the pulmonary tissue, the substance of the heart, the mucous membrane of the stomach, the mucous membrane and submucous connective tissue of the large intestines. These extravasations had a common origin in the morbid state of the blood. Moreover, the disordered appearance which the spleen presented, and its possible connection with the blood-lesion, should not be overlooked. That organ was enlarged, softened, full of blood, and its parenchyma had a dark-brown, rotten look.

It is also worthy of remark that both the substance and the membranes of the brain were paler than natural, or anæmic. This exsanguinated condition was probably due, in great part at least, to a compression of the cerebral blood-vessels, but more especially of the cerebral capillaries, which was exerted by the extravasated blood.

No symptoms of hemiplegia were observed. This was due to the fact that the effusions were poured into the cavity of the arachnoid and upon or over both cerebral hemispheres.

CASE LII.

Cachexia, convulsions with great depression, purpura hemorrhagica, and speedy death ; autopsy ; extensive meningeal hemorrhage ; cerebral anæmia, etc.

A little boy, five weeks old, the child of healthy parents, was brought to Dr. West. He had been perfectly well for the first fortnight after birth; he then, without apparent cause, grew drowsy, vomited often, and got a jaundiced appearance. His abdomen became large and hard, and he cried when pressure was made on the right hypochondrium; these symptoms continued when Dr. West saw him. A leech was applied to his right side, his bowels, previously constipated, were acted on by small doses of calomel and castor-oil, and in three days he lost the yellow tinge of his skin, became cheerful, and seemed much better. But he was now, July 18th, suddenly seized with hurried respiration and great depression, soon followed by violent convulsions, during which he screamed aloud. At the same time it was observed that his left hand had begun to swell, and to put on a livid hue, and on the 20th the right hand also became œdematous. His whole surface grew quite sallow, the œdema and livor of his left hand increased, and there were small spots of extravasated blood over each knuckle observed on the 20th. The right elbow was slightly livid, the right hand much swelled, but natural in color, and a small black spot had appeared under the chin corresponding to the knot of the cap-string. The fits recurred very frequently, the child in the intervals lying quite still; the pupils were contracted, and his condition seemed to be one of extreme exhaustion rather than of coma. He lost the power of swallowing on the 20th, and, after several returns of less violent convulsions, died at 9 A.M., July 21st, about sixty hours after the first fit occurred.

The sinuses of the dura mater were full of fluid blood; a black coagulum, three or four lines thick, covered the whole posterior part of both hemispheres, extending from the posterior third of the parietal bones, occupying the whole concha of the occipital bone, and reaching along the base of the skull to the foramen magnum. A little blood was also effused about the anterior part of the cerebral base, but the quantity was very small in comparison with what was found at the posterior part. The substance of the brain was very pale; and all the organs were anæmic, except the liver, which was gorged with fluid blood, while the heart was quite empty. The ductus arteriosus was closed, the foramen ovale admitted a probe with ease, the ductus venosus admitted one with difficulty. (Vide *West's Lectures on Diseases of Infancy and Childhood*, pp. 65, 66, Am. ed. 1868.)

Dr. West relates another case in which passive hemorrhage into the arachnoid occurred in a child exhausted by long-continued illness, aggravated by poverty and want. This child had been under his care from the age of two to that of five months, in consequence of frequent attacks of hæmatemesis and purging of blood; and, though he improved, he never became strong, his motions being almost always white and deficient in bile. After he was weaned, the coarse fare to which he was restricted by indigence did not nourish him; he lost flesh and strength, and, when almost three years old, was puny and emaciated. Three days before death, an attack of diarrhœa occurred, and induced great exhaustion. While suffering from this affection, he suddenly grew comatose, cold, and almost pulseless, and his breathing became so slow that he inspired only four or five times per minute. He lay in this state for twenty-four hours, and then quietly died.

Nearly six ounces of dark coagulated blood were found in the sac of the arachnoid, over the right hemisphere of the cerebrum; a little blood was also effused beneath the arachnoid, and there was a very small clot in the lower and front part of the middle lobe of the same hemisphere, but no ruptured vessels could be perceived. Great anæmia of every organ, and extreme attenuation of the walls of the heart, were the only other remarkable appearances. (Vide *op. cit.* p. 66.)

Comments.—Of these two cases of meningeal hemorrhage related by Dr. West, the former (Case LII.) had cutaneous or subcutaneous extravasation of blood in several different parts, and, therefore, belongs to the same category as Case LI., in which purpura hemorrhagica occurred; and the comments which we made on that case are equally applicable to this one.

In the latter of these two cases the extravasation of blood upon the brain was also due, in all probability, to a morbid state of the blood itself, since this child was exhausted by long-continued illness, aggravated by poverty and want.

Mushet mentions a case of cerebral hemorrhage similar to the foregoing. It occurred in a female child, aged two and a half years, "of scrofulous constitution, always fed upon very poor diet, consisting principally of oatmeal, and for many months subject to great functional disorder of the abdominal viscera." After death, a large quantity of blood was found effused upon the surface of the brain. The liver, spleen, kidneys, and mesenteric glands were unusually large. The meningeal hemorrhage in this case, also, was doubtless due to a deteriorated condition of the blood. (Vide *A Practical Treatise on Apoplexy, (cerebral hemorrhage,)* pp. 63, 64; also Case of Sanguineous Apoplexy in *London Med. and Phys. Journal*, No. 47, for 1822, by T. H. Greenhow, Esq., of Newcastle.)

Mushet mentions another case of meningeal hemorrhage which had a cachectic origin. The subject was a little girl two years and seven months old. There was effusion into the cavity of the arachnoid and at the base of the brain, purpuric and passive in character. The case was originally reported by Dr. Richard Quain in the *London Journal of Medicine* for January, 1849.

Abercrombie's case (No. CXX.) was an instance of cerebro-spinal hemorrhage occurring in a boy nine years old. He had convulsions, and the symptoms of coma were gradually developed. There was copious extravasation of blood. It filled all the ventricles, and extended along the whole course of the spinal cord. (Vide *Abercrombie on the Brain*, etc., pp. 237, 238.)

But hemorrhage into the substance of the brain does sometimes occur in infancy and childhood, although, as already stated, the arachnoid is almost always the seat of cerebral hemorrhage at this time of life. For example, Billard gives the case of an infant only three days old, and apparently quite healthy, who suddenly presented the symptoms of well-marked apoplexy, and died; a clot of blood was found in the substance of the left hemisphere of the brain, immediately outside the corpus striatum. Serres saw a similar case in a child three months old. (Vide *Aitken's Science and Practice of Medicine*, vol. ii. p. 322.) Wythes has recorded three cases of apoplexy occurring in young children who previously had been in good health, except a pain about the left instep of the first. There was effusion of blood within the skull in all three, and marked softening of the brain in one. (Vide *New Sydenham Soc. Year-Book* for 1859, pp. 179, 184.) Dr. Richard Quain has reported an interesting example, occurring in a boy aged nine years, which was characterized by coma, convulsions, paralysis, and death in seven hours from the time of seizure, previous to which he was in the enjoyment of good health. After death, a large clot of blood was found in the right hemisphere of the brain, and the left ventricle of the heart was much hypertrophied. (Vide *Tanner on Diseases of Infancy and Childhood*, p. 218; also the *London Journal of Medicine* for January, 1849.)

Authorities on the subject, however, generally concur in stating that hemorrhage into the substance of the brain is a very rare occurrence in infancy and childhood. Dr. West remarks that he has only twice met with distinct extravasation of blood into the substance of the brain in children. (Vide *Lectures on the Diseases of Infancy and Childhood*, p. 67.) These two cases of Dr. West we shall now proceed to relate.

Case LIII.

Vomiting, headache, stupor, convulsions, and partial hemiplegia; death finally occurred in convulsions; autopsy; extensive hemorrhage in the cerebral substance and beneath the arachnoid.

The patient was a girl, aged eleven years, born of healthy parents, whose own health had been quite good till she was six years old. Then the extraction of a molar tooth was followed by necrosis of a large part of the lower jaw, and by the formation of abscesses in the face and head, from which bone escaped. An abscess attended with similar exfoliation of bone formed on her right foot, and it was three years before she completely recovered. Though much disfigured, her health continued good until April 12th. She was then suddenly, and without known cause, attacked with vomiting and headache, for which no treatment was adopted for ten days, except the occasional administration of an aperient. During this time, however, a state of stupor gradually stole over the child, for which, on April 21st, a blister was applied to the back of her neck, with great relief. On April 23d she had two attacks of convulsions, with an interval of four hours between them. She struggled much during their continuance, especially with her right side; when they subsided, partial palsy of her left side remained; she complained much of her head, and from time to time sank into a state of stupor, from which, however, she could always be roused. Very free purgation on April 24th, and the application of another blister to the nape of her neck, were followed by some amendment. On the evening of the 25th, another fit occurred, with symptoms similar to those observed on the previous occasions; but it was not followed by any increase in the palsy of the left side; nor was the degree of stupor so great as on the former occasion. Mercurials, which had been employed from the commencement of the attack, now produced a decided impression on her mouth, and the abundant action of her bowels was again succeeded by much improvement in her condition. Her pulse, which had varied from sixty to seventy, now continued about seventy, and was natural; and she improved without taking any medicine, except occasional aperients. The headache occasionally returned, but each time it was less severe than the time before. On the evening of May 15th, however, she was suddenly attacked with severe pain in the abdomen, which was soon followed by convulsions and coma, and in sixteen hours she died convulsed, on the thirty-sixth day from the first occurrence of pain in her head.

On opening the head, blood was found effused into the subarachnoid tissue over a great part of the right cerebral hemisphere. The quantity of blood, however, was nowhere very considerable, but merely occupied the sulci between the convolutions. A large clot, rather larger than a hen's egg, but more irregular in shape, was found in the substance of the right hemisphere, on a level with, and just exterior to, the lateral ventricle. This extravasation was perfectly black throughout, the coloring particles of the blood being equally diffused through it, and no appearance betokened that hemorrhage had previously occurred. The anterior cerebral artery ran for a considerable distance just outside the clot, but it could not be shown that it had given way at any point. The brain-substance surrounding this clot was softened. (Vide *West's Lectures on Diseases of Infancy and Childhood*, pp. 67, 68, Am. ed. 1868.)

Comments.—This is the first case of cerebral hemorrhage occurring in childhood, that we have related, wherein it is distinctly mentioned that hemiplegia was present, except, perhaps, the boy reported by Dr. Richard Quain, in whom a large clot of blood was found in the right hemisphere of the brain after death; for it is stated that paralysis was present in his case, but the form thereof is not given. As in adults, extravasation of blood into the arachnoid cavity generally does not produce the symptoms of hemiplegia, so when cerebral hemorrhage occurs in infants and children, hemiplegia is not induced, unless a clot of considerable size has been formed in the substance of the brain.

In the case just related, the extravasation into the substance of the right hemisphere was accompanied by considerable subarachnoid effusion, that is, by effusion into the meshes of the pia mater; so that meningeal hemorrhage, after all, was not entirely absent.

Case LIV.

Scarlatina, anasarca, chronic peritonitis and pleurisy; death apparently from syncope; autopsy; thrombosis of the sinuses of the dura mater; four clots of effused blood in the brain-substance, etc.

A healthy little girl took scarlatina when eight months old. After it, she did not regain her health, but remained restless and feverish; and her eyelids were often slightly swelled. When she was ten and a half months old, her mother noticed, in addition to the puffiness of the eyelids, a swelling of the legs and abdomen, for which she came under Dr. West's care at the age of eleven months. The legs were then very œdematous, and fluctuation was distinctly felt through the walls of the abdomen; urine scanty and high-colored. In the course of the next three weeks she improved considerably; urine much increased, anasarca much diminished, and abdomen one and a half inches less in circumference. A fit of convulsions now occurred without apparent cause; no other symptoms of cerebral mischief followed, and they did not return. After the lapse of another week, a discharge of sero-purulent fluid from the umbilicus burst forth, and continued for several days in quantities ranging from four to eight ounces per day. This discharge was attended with some improvement in the child's health; but after it had continued for eleven days, fever and dyspnœa suddenly came on, with dulness on percussion over the right side of chest, and absence of the respiratory murmur in that situation. The discharge ceased when the thoracic symptoms occurred, but returned at the end of a week, though scantily. The child now grew thinner and weaker, and soon sank into a hectic state. No new symptoms occurred, till she was suddenly seized with extreme faintness, amounting almost to perfect syncope. She rallied, however, under the use of stimulants; but forty-eight hours afterward the faintness returned, and terminated in death, without any convulsive movements, just five and a half months after the attack of scarlatina and two months after coming under Dr. West's care.

The *Autopsy* revealed pleurisy of the right side with about six ounces of pus in that pleural cavity, and peritonitis with three pounds of pus in the abdomen—the passage being still traceable through which the fluid had escaped at the umbilicus.

The dura mater adhered firmly to the skull, along the posterior half of the longitudinal sinus, at the torcular Herophili, and along the left lateral sinus; but elsewhere it was easily detached from the cranium. The sinuses on the right side were healthy, but the blood within them was almost entirely coagulated. The posterior half of the longitudinal sinus, the torcular, the left lateral, and left occipital sinuses were blocked up with a fibrinous coagulum, precisely such as one sees in inflamed veins, and the clot extended into the internal jugular vein. The coats of the longitudinal, and of the inner half of the left lateral sinus were much thickened, and their lining membrane had lost its polish, was uneven, and presented a dirty appearance.

There was some congestion of the arachnoid, a considerable quantity of fluid in the ventricles, and sections of the brain presented more bloody points than natural, especially on the left side. The base of the brain was perfectly healthy on the right side, but there was great venous congestion on the left side beneath the middle lobe of the cerebrum; the cerebral veins in that situation were distended with coagula, and their coats were thickened. Toward the anterior part of the left middle lobe were four *hemorrhagic effusions*, in all of which the blood retained its natural color. Each of these effusions was connected with an obstructed and distended vein. The largest clot extended an inch into the substance of the brain; the others were of smaller extent. (Vide *West's Lectures on Diseases of Infancy and Childhood*, pp. 107–109, Am. ed. 1868.)

Comments.—In this case *thrombosis of the cerebral sinuses* occurred, and the several extravasations of blood into the substance of the brain were obviously due to a mechanical cause, namely, to occlusion of certain sinuses of the dura mater, and of certain veins which empty into them, with coagulated blood. Thus, the circulating blood, unable to find its way back toward the heart through the natural venous channels, burst through the walls of the distended capillaries, and escaped into the substance of the brain. The hemorrhage was therefore essentially venous and passive in character.

CASE LV.

Diarrhœa and vomiting, followed by collapse, head-symptoms, cyanosis of the face, and death in three days; autopsy; thrombosis of the sinuses of the dura mater; extensive hemorrhage into the cerebrum and meninges.

A boy, three weeks old, and artificially fed, was seized with diarrhœa and vomiting. Fontanelle depressed. Collapse, sopor, strabismus, paralysis of the right facial nerve. Muscles of the nape of neck and extremities contracted. Right jugular vein not quite so full as the left. Later, pulselessness and cyanosis of the face; the fontanelle fills up again, and becomes tense; both jugulars alike imperfectly filled. Superficial veins of right ear much distended. Death in three days.

Autopsy.—Body showed signs of marasmus. Fontanelle and sutures moderately open and tense. In the superior longitudinal sinus a very abundant blackish-red coagulum, partially adherent, and extending for some distance into the lateral sinuses. Pia mater and brain anæmic; substance of the latter soft. In the right cerebral hemisphere there was a cavity with ragged walls which occupied nearly its whole extent, and encroached slightly upon the left hemisphere. It contained a large clot of blood. At the base of the cerebellum also rather considerable extravasations were found in the course of the vessels of the pia mater. In the veins of that locality black rather firm coagula were found at different points. In the mitral and tricuspid valves there were abundant gelatinous thickenings; upon one fold of the latter, and in the outer coat of the ascending aorta, slight recent extravasation. Lungs inflated and somewhat œdematous. Spleen small. Lithic acid infarcta in kidneys. Catarrh of intestinal canal. (*Dr. Gerhardt's case.* Vide *Dusch on Thrombosis of the Cerebral Sinuses*, pp. 113, 114, New Sydenham Soc. translation.)

Comments.—This case furnishes us with another instance in which cerebral hemorrhage was produced in a young child by the spontaneous plugging of some of the cerebral sinuses and some of the cerebral veins with coagulated blood. The mechanical obstruction to the circulation thus effected led to engorgement of the vessels which emptied into those that were occluded, and finally to the escape of their contents into the substance of the right cerebral hemisphere, and into the arachnoid at the base of the cerebellum. This case, then, affords another example of infantile cerebral hemorrhage, which was venous in character and mechanical in point of origin.

Case LVI.

Typhoid fever, convulsions, purpura hemorrhagica, and death; autopsy; thrombosis of superior longitudinal sinus; very extensive meningeal hemorrhage; blood extravasated also into the substance of the right cerebral hemisphere.

A girl, twelve years old, had been suffering for a month with feverish symptoms, anorexia, profuse diarrhœa, etc. When admitted into the hospital, her case was looked upon as an advanced stage of typhus. The diarrhœa continued, and her condition at the end of thirteen days was little improved. Clonic and tonic convulsions, with loss of consciousness, but not of sensibility, suddenly supervened. These phenomena lasted an hour, after which the patient became quiet. A fresh accession soon occurred, however, with trembling of the muscles, which lasted till death. The convulsions were followed by coma, with contraction of the fore-arms and hands, and contracted pupils; pulse 100; death.

Autopsy.—There were found typhoid ulcers, partly cicatrized, partly undergoing the healing process; and small spots of purpura on the mucous membrane of the stomach and pelvis of the kidneys; similar small ecchymoses formed beneath the skin of the lower extremities, in the last hours of life. The whole of the longitudinal sinus was obstructed by a coagulum everywhere adherent and partly discolored, as also all the veins of the pia mater communicating with it. The other sinuses were unchanged. The whole of the pia mater on the surface of the brain and between its convolutions replete with [effused] blood, and in some places, especially in the right Sylvian fissure, more considerable extravasations existed. A cavity filled with blood, partly recent, partly of longer standing, was found in the anterior lobe of the right cerebral hemisphere. (*Bouchut*, quoted by *Dusch.* Vide *Thrombosis of the Cerebral Sinuses*, p. 111.)

Comments.—The history of the foregoing case possesses more than ordinary interest because it shows us how typhoid fever sometimes terminates in death with unusual symptoms that are referrible to the brain, and in a thoroughly exceptional manner. These head-symptoms were sudden loss of consciousness, but not of sensibility, with convulsions or spasms both clonic and tonic in character, trembling of the muscles, coma or complete suspension of the cerebral functions, and death. The autopsy showed that blood had been extravasated into the substance of the right cerebral hemisphere, and into the subarachnoid connective tissue or meshes of the pia mater on the whole surface of the cerebrum, in consequence of the great longitudinal sinus of the dura mater having become occluded throughout its whole extent with clotted blood. This case, therefore, is an instance of cerebral thrombosis terminating in cerebral hemorrhage, and belongs to the same category as Cases LIV. and LV.

It is also worthy of remark that spots of purpura hemorrhagica presented themselves during the last hours of life in this case. This circumstance shows that there was a morbid state of the blood present, which favored the occurrence of hemorrhage; and thus we find that the thrombosis of the longitudinal sinus was assisted in producing the cerebral hemorrhage by the morbid state of the blood itself.

We shall next present a brief sketch of three additional cases in which cerebral hemorrhage was caused by thrombosis of the cerebral sinuses in little children. These extravasations of blood into the substance, or on the surface, of the brain, were all venous and passive in character, and mainly due to mechanical obstruction of the cerebral circulation.

1. A girl, two years old, had freely suppurating eczema of the hairy scalp. The discharge suddenly ceased, cerebral symptoms, indicating an affection of the left hemisphere, showed themselves, and terminated in death.

Autopsy.—In the posterior part of the longitudinal sinus was found a plug broken down by suppuration, the walls of the sinus were thickened, and the veins communicating with it were seen to be filled with stiff thrombi. There was an *extravasation of blood* beneath the arachnoid on the left side. (*Tonnelé*, quoted by *Dusch.* Vide *Thrombosis of the Cerebral Sinuses*, pp. 96, 97, New Sydenham Soc. translation.)

2. A child, two years of age, which had long been very weakly, died suddenly with symptoms of suffocation. At the *autopsy*, a fresh clot was

found in the anterior part of the superior longitudinal sinus, while the posterior part of this sinus was filled with false membranes and a fluid resembling the lees of wine; the veins communicating with this sinus were much distended with blood. There was a large *hemorrhagic clot* in the centre of the right hemisphere. (*Tonnelé*, quoted by *Dusch.* Vide *Thrombosis of the Cerebral Sinuses*, p. 106.)

3. A little girl, four years old, having ophthalmia and swelling of the glands, was seized with pneumonia of the upper lobe of the right lung. She had swelling of the submaxillary gland and dilatation of the pupils. Death occurred on the fifth day.

Autopsy.—In the superior longitudinal sinus was a clot which was closely adherent to the walls of the sinus, and partly in a state of purulent softening in its interior. This clot extended for some distance into both lateral sinuses. All the cerebral veins opening into these sinuses contained pus mixed with clots. An *ecchymosis* was visible on the left hemisphere. The brain could not be examined more minutely. (*Cruveilhier*, quoted by *Dusch.* Vide *Thrombosis of the Cerebral Sinuses*, p. 107, New Sydenham Soc. translation.) Inasmuch as an ecchymosis was visible on the left hemisphere, while the longitudinal sinus was thoroughly occluded with coagula, it is not improbable that, if the examination of the brain could have been made complete, well-marked cerebral hemorrhage would have been found in this case.

A few words concerning thrombosis of the cerebral sinuses may not be amiss in this place. It is an affection which essentially consists in the spontaneous coagulation of the blood in the sinuses of the dura mater. It does not occur in healthy subjects. It is met with only in the cachectic, and those who are exhausted by long-continued disease; that is, in subjects whose blood has become disordered, having, among other changes, acquired an abnormal tendency to coagulate. In occasional instances, thrombosis is produced by inflammation of the sinuses themselves. But this is not the rule; it is only the exception. In almost every case it is a morbid state of the blood which is the main cause of thrombosis of the sinuses of the dura mater.

Etiology of Infantile Cerebral Hemorrhage.—Dr. West remarks that effusion of blood upon the brain occurs much oftener in weakly children than in robust ones, and that there is reason for supposing the hemorrhage to be passive in character and dependent on an altered state of the blood in many of the cases. He considers such cases to constitute a cachectic form of cerebral hemorrhage. Our own experience and researches sustain Dr. West's views. We have found that extravasation of blood into or upon the brain occurs much more frequently in cachectic children than in healthy ones. For example, we have related in this chapter, with more or less fulness of detail, fifteen cases in which infantile cerebral hemorrhage took place, exclusive of Cruveilhier's seven cases of so-called apoplexy in the newly-born, and Wythes' three cases, in which the seat of the effusion is not specified. Of these fifteen cases only three were in good health when the attack occurred, while twelve were in a cachectic state. Three or four of them had purpura hemorrhagica, and six thrombosis of the sinuses of the dura mater; so that the morbid state of the blood and the passive character of the hemorrhage were very well marked in most of them.

First among the causes of cerebral hemorrhage occurring in cachectic children must be ranked thrombosis of the sinuses of the dura mater. In such cases effusion of blood occurs, not because the walls of the minute arteries and capillaries of the brain have become weakened by disease, but because the flow of blood from a large part of the brain has been arrested by a mechanical obstacle, namely, plugging the venous sinuses with coagula.

Next among the causes of cachectic cerebral hemorrhage occurring in children, purpura hemorrhagica must be mentioned. In the purpuric cases, also, the extravasation does not occur in consequence of any disease of the cerebral blood-vessels, but because the blood itself has become changed, and acquired an abnormal tenuity. Besides, substantially the same remarks hold good in regard to the other instances of cachectic cerebral hemorrhage which are met with in infancy and childhood.

We have mentioned that in three of the fifteen cases which we have presented in this chapter, the subjects were in good health when the attack occurred. This statement leads us to briefly mention the so-called idiopathic or primary form of infantile cerebral hemorrhage. Like the corresponding disease in adults, it, for the most part, depends upon disease of the small blood-vessels of the brain, as we have already shown in Chapter V. It is also a disorder which very rarely occurs in young children. Henoch, however, relates three cases which presented themselves in children aged one and a half, seven, and seven and a half years. All three were suddenly seized in the midst of undisturbed health with hemiplegia, which in two passed into pricking sensations and numbness of the affected hand, and epileptic convulsions. The convulsive phenomena were exclusively on that half of the face and body which on their cessation remained paralyzed. In the child aged seven and a half years all convulsive phenomena were wanting. (Vide *New Sydenham Soc. Retrospect* for 1867–8, p. 427; also *Beiträge zur Kinderheilk.* 1868.)

The causes of the cerebral hemorrhage of new-born infants are, for the most part, mechanical in their nature and operation. They have already been considered at sufficient length.

Symptoms of Infantile Cerebral Hemorrhage.—The term infantile apoplexy, as we have elsewhere stated, is not synonymous with infantile cerebral hemorrhage, for apoplectic stupor often occurs in children when there is no extravasation of blood in the brain or its membranes, and the latter is not always nor even generally attended with the symptoms of the former, in the etymologically correct sense of the word.

"All writers, even those who, like MM. Rilliet and Barthez, have thrown most light on the anatomy and pathology of cerebral hemorrhage in the child, concur in representing its *symptoms* as extremely obscure. Paralysis, which, in the grown person, is one of the most frequent results of the escape of blood from the cerebral vessels, is so rare in the child that it was observed by M. Legendre only in one out of nine cases, and by MM. Rilliet and Barthez in one out of seventeen cases." (Vide *West's Lectures on the Diseases of Infancy and Childhood*, p. 64, Am. ed. 1868.) Paralysis or hemiplegia is mentioned as having occurred in but two of the fifteen cases of infantile cerebral hemorrhage which we have presented in this chapter. Dr. West thinks that this peculiarity of cerebral hemorrhage occurring in infants is mainly due to the fact that in these cases the blood is generally extravasated into the cavity of the arachnoid, so that the pressure which it exerts on the brain is widely diffused over the surface of the organ, and is nowhere very considerable. In the fifteen cases of cerebral hemorrhage occurring in infancy and childhood which we have presented, it is noted that the blood was effused into the meninges in *seven* cases, into the meninges and brain-substance in *three* cases, and into the brain-substance alone in *five* cases, yet the occurrence of paralysis or hemiplegia was remarked in only two cases. It is probable that the state of the fontanelles and sutures as to openness and flexibility has a good deal to do with the presence or absence of the symptoms of hemiplegia in the children who get sanguineous extravasation in the substance of the brain; for, when the fontanelles and sutures are open and yielding, it is obvious that a considerable quantity of blood may be effused into the cerebrum without inducing such a degree of compression as would close the cerebral capillaries and occasion anæmia and consequent arrest of function of any considerable part of the cerebral substance. But, when hemiplegia occurs in infants it should not be looked upon as undoubted evidence that cerebral hemorrhage has occurred, for Henoch says that, of all the numerous cases of hemiplegia which he has seen in children, in those that came to post-mortem examination tuberculosis of the brain was found to be the cause, again establishing the enormous preponderance of this disease over all other forms of alteration of brain-structure in childhood. (Vide *New Sydenham Soc. Retrospect*, 1867–8, p. 427; also *Beiträje zur Kinderheilk*, 1868.

Dr. West further remarks concerning the symptoms of infantile cerebral hemorrhage: "The absence of paralytic symptoms, however, is not the sole cause of the obscurity of these cases, but the indications of cerebral disturbance, by which they are attended, vary greatly in kind as well as in degree. The sudden occurrence of violent convulsions, and their frequent return, alternating with spasmodic contraction of the fingers and toes in the intervals, appear to be the most frequent indications of the effusion of blood upon the surface of the brain. I need not say, however, that such symptoms taken alone would by no means justify you in inferring that effusion of blood had taken place. Many circumstances having reference to the previous history of the child, as well as to its present condition, must be taken into account in forming a diagnosis. Hemorrhage into the arachnoid cavity is most frequent in early childhood—symptoms such as have been enumerated would therefore acquire additional diagnostic importance in proportion to the tender age of the child in whom they occurred. The probability of their betokening this accident would be still further strengthened if the child who experienced them had previously suffered from frequent attacks of cerebral congestion, or had been recently exposed to the sun without proper covering for the head; or had been placed in other circumstances calculated to favor determination of blood to the head." (Vide *Lectures on the Diseases of Infancy and Childhood*, p. 65, Am. ed. 1868.)

Treatment of Infantile Cerebral Hemorrhage.—On this topic there is not much to be said. The indications are to remove the cause of the hemorrhage, and to obviate the tendency to death. So far as the causal indications are concerned, we are, in most cases, quite powerless to do good. For example, if the extravasation be due to thrombosis of the sinuses of the dura mater, as it often is, we do not possess any means of dissolving the coagula in these sinuses, and of setting the blood to flow again in its natural channels from the substance of the brain toward the heart. Or, again, if the effusion be occasioned by purpura hemorrhagica, as it not unfrequently is, we are utterly unable to restore its lost plasticity to the disordered blood; and, indeed, in most cases, where the cerebral extravasation is due to a cachectic state of the system, we are almost equally powerless to remove the cause of the bleeding after it has actually begun. Prior to this event, however, much may be done, in some cases at least, by adopting such a plan of treatment as shall prevent or remove the cachexia itself.

But there is one class of young children exposed or liable to the occurrence of cerebral hemorrhage in whom we can sometimes do a great deal in the way of removing its cause, namely, the cases in which determination of blood to the head, fluxionary hyperæmia or active congestion of the brain, as it is often called, threatens to produce or is actually the cause of sanguineous effusion within or upon the brain. In such cases we should combat the tendency to cerebral congestion by venesection, by leeches applied to the head, by the administration of purgatives, and by cold affusion of, or even the application of the ice-bag, to the head, according to the age and strength of the little patient, and according to the effects produced. The effects of cold affusion and of the ice-bag should, however, always be carefully watched.

We can do something to obviate the tendency to death, at least in occasional instances, by attending to the alimentation of the patient, that is, by seeing that food enough to support life gets into his stomach at proper intervals, by the administration of alcoholic stimulants in asthenic cases to keep the heart beating and the blood in circulation, by the use of stimulating enemata, and by the application of epispastics to rouse the flagging energies of the nervous system.

We have elsewhere pointed out how the so-called apoplexy of the newly-born may, not unfrequently, be prevented by the timely completion of the process of labor by artificial means, such as the employment of forceps, etc. When the infant is born asphyxiated, the various measures for resuscitation which are usually recommended in works on obstetrics should be employed. Cruveilhier, however, thinks that, in cases where the fœtus is healthy at the commencement of labor, it can be saved by the opportune termination of the labor by artificial means, in the majority of instances, when otherwise it must perish of asphyxia; and on this proceeding we should place our chief reliance against this accident.

CHAPTER IX.

ON PULMONARY APOPLEXY, (SO-CALLED,) OR PULMONARY EXTRAVASATION, AND HEMORRHAGIC INFARCTION OF THE LUNGS, ETC.

Two varieties of pulmonary apoplexy recognized.—But neither of them is attended with the phenomena which characterize apoplexy in the correct sense of the term, except in very rare instances.—To call this affection apoplexy is unphilosophical and absurd.—Remarks of Drs. Trousseau and Gendrin on this point.—The term pulmonary apoplexy has been dropped by the Royal College of Physicians at London, and the term *Pulmonary Extravasation* adopted in its stead.—The new term is quite unobjectionable.—There are two varieties of pulmonary extravasation: 1. The capillary, or circumscribed. 2. The lacerated, or diffused. The capillary variety may be produced, *a*, by diseases of the blood, for example, purpura hemorrhagica, and, *b*, by the lodgment of embolia in the minute branches of the pulmonary artery.—The last species is of frequent occurrence, great practical importance, and has been described under the name of hemorrhagic infarction of the lungs.—*Etiology of Pulmonary Hemorrhagic Infarction.*—It consists in a capillary hemorrhage, confined to a small and sharply-defined section of the lung, and often bounded by the limits of a single lobule.—It is produced by the plugging of small branches of the pulmonary artery with embolia, which are usually brought from some remote part of the body.—The credit of this valuable discovery is due to Virchow.—Embolia often originate in disintegrating thromboses of peripheral veins.—The embolia, which so often produce hemorrhagic infarction of the lungs in cases of heart-disease, come from the right chambers of the heart itself.—The process by which capillary hemorrhage is produced in these cases of pulmonary embolism.—Ludwig's explanation of it.—*Anatomical Appearances produced by Pulmonary Hemorrhagic Infarction.*—Those which result from disease of the heart vary in size from that of a hazel-nut to that of a hen's egg, are blackish-red or almost black in color, completely inelastic, and void of air, so that they feel from without like hard knots.—Old infarctions look paler and yellowish.—Those which are produced by embolia derived from the peripheral veins have generally a small size, (varying from that of a pea to a cherry,) a cuneiform shape, and a superficial situation.—Metastatic pneumonia or abscesses, the result of infarctions, described.—*Symptoms and Course of Pulmonary Hemorrhagic Infarction.*—The symptoms mentioned in detail.—Symptoms of septicæmia or pyæmia, how produced in cases of hemorrhagic infarction of the lungs.—*Treatment of Pulmonary Hemorrhagic Infarction.*—It must be principally directed to the relief of prominent symptoms.—When abstraction of blood should be employed; stimulants administered, and epispastics applied, etc.—*On the Second Variety of Pulmonary Extravasation*, namely, that in which the blood is effused from vessels of some considerable size, the pulmonary tissue is more or less torn or broken down by the extravasation itself, and cavities filled with blood of corresponding size, are formed in the pulmonary parenchyma.—*Etiology.—Anatomical Appearances.—Symptoms.*—The disorder is absolutely deadly and not susceptible of treatment.—The so-called apoplexy of the liver, spleen, kidneys, etc.,

consists in the extravasation of blood into the substance of these organs.—Such a use of the term apoplexy is also inappropriate and should be abandoned.—They can be designated as renal, splenic, or hepatic extravasations, according to the organ affected by the hemorrhage, with perfect propriety.

The term pulmonary apoplexy has been applied to a disorder of the lungs, which consists in the extravasation of blood into the pulmonary tissue, or parenchyma. It is also a disorder whose presence has not been recognized during life in most of the cases where it has occurred. Generally, it has not been discovered until the post-mortem examination revealed it. *Two varieties* of pulmonary apoplexy have been admitted: 1. The *capillary* or *circumscribed*, that is, a variety of pulmonary extravasation in which the blood is, for the most part, effused from the ultimate or capillary branches of the pulmonary artery only, in which also the pulmonary tissue is not lacerated, and hemorrhagic cavities in the pulmonary substance are not formed. 2. The *lacerated* or *diffused*, that is, a variety in which the blood is extravasated from branches of the pulmonary artery, or from other thoracic blood-vessels, having considerable size, in which also the pulmonary tissue is more or less torn or broken down by the extravasation itself, and in which cavities filled with blood, of corresponding size, are formed in the pulmonary parenchyma. Thus, it becomes apparent that each of these varieties of pulmonary apoplexy is quite distinct from the other, and possesses characteristic features.

Now, when we come to investigate the symptoms or phenomena which belong to the so-called apoplexy of the lungs, we find that it is never, except in very rare instances, attended with sudden loss of consciousness, and volition, and sensibility, and motility, nor with the sudden development of coma, nor with any of the phenomena which characterize apoplexy in the ancient and clinical sense of the term. It therefore seems quite absurd to use the word apoplexy to designate a parenchymatous hemorrhage occurring in the lungs. To say the least, such an application of the term is arbitrary, inappropriate, and unphilosophical. It is clear, then, that this employment of the term apoplexy should be dropped altogether, and that another and more appropriate name for this affection of the lungs should be adopted and brought into general use.

On this point Trousseau justly observes: "The affection, in fact, has no characters in common with cerebral apoplexy, with which some wish to compare it; the term *apoplexy* always implies the idea of sudden seizure and active congestion, characters belonging much more to bronchial than to pulmonary hemorrhage, which latter is usually more or less passive. It is true that cases have been recorded of true pulmonary apoplexy occasioning sudden death, and presenting at the autopsy more or less extensive effusion of blood into the middle of a lacerated lung, presenting very nearly the same appearance as cerebral tissue into which there has been violent hemorrhage. *Apoplexy* is a term which would be much more applicable to active congestion of the lung, a disease which is not very uncommon, but which is very seldom accompanied by sanguineous effusion which can be properly called a hemorrhage. Dr. Gendrin has substituted for pulmonary apoplexy the term '*pneumo-hêmorrhagie*,' which succinctly expresses, without any ambiguity, *extravasation of blood into the tissue of the lungs.* (Vide *Traité de Médecine Pratique*, t. i. p. 638.) He rejects the term 'apoplexy,' because the invasion of the disease is seldom sudden, and is not accompanied by rapidly dangerous symptoms like those of cerebral apoplectic seizures; because the alterations of tissue differ in many respects from the alterations of tissue produced by encephalic hemorrhage, and because, in a word, it does not embrace all the forms and degrees of the pathological state in question." (Vide *Trousseau's Lectures on Clinical Medicine*, vol. iii. p. 148, New Sydenham Society translation.)

The term pulmonary apoplexy has also been dropped from the Revised Nomenclature of Diseases issued by the Royal College of Physicians of London, in 1869, and the term *pulmonary extravasation* has been adopted in its stead. The term pulmonary hemorrhage would not answer as a substitute, because it is generally considered to be synonymous with hæmoptysis. This change, then, for reasons stated above, was eminently fit to be made, and must prove useful in a scientific point of view. The first object which the author had in mind when he planned the outline of this chapter, was to state some reasons why this change should be made, and thus to influence others in favor of its adoption.

We shall now proceed to give a brief account of pulmonary extravasation, or hemorrhage into the parenchyma of the lungs, as a substantive disorder. As already intimated, there are two varieties of this affection which are quite distinct from each other in respect to origin, anatomical relations, course, and consequences. In one of them, the blood is effused from the capillaries or ultimate branches of the pulmonary artery, the pulmonary tissue is not lacerated, and no hemorrhagic foci are formed. In the other, the blood is extravasated from vessels of some considerable size, the pulmonary tissue is more or less torn or broken down by the extravasation itself, and cavities filled with blood, of corresponding size, are formed in the pulmonary parenchyma.

Of these two varieties the first mentioned occurs much oftener than the other. Its practical importance is also much greater than that of the other. It is for the most part occasioned by the lodgment of embolia in the minute subdivisions of the pulmonary artery. In occasional instances, however, it is produced by a morbid state of the blood itself, such, for example, as we find in the disease called purpura hemorrhagica. This form of capillary hemorrhage into the pulmonary tissue was found at the autopsy in Case LI.[1] The spots of extravasation in that case were as large as peas. But purpuric hemorrhage into the parenchyma of the lungs is of comparatively infrequent occurrence, and of importance in a pathological, rather than in a practical, point of view. It, therefore, claims only a brief notice in this place. Not so, however, with the form of capillary hemorrhage into the pulmonary tissue, which is produced by small embolia lodged in the minute branches of the pulmonary artery. It is of frequent occurrence, and of great practical importance. It has been described by Virchow, Niemeyer, and others, under the title of *hemorrhagic infarction of the lungs.* We do not perceive any valid objection to this name, and therefore shall continue to use it.

Etiology of Pulmonary Hemorrhagic Infarction.—It consists in a capillary hemorrhage, confined to a small and sharply-defined section of the lung, and often bounded by the limits of a single lobule. The blood is effused, partly within the cavity of the air-vesicles and terminal bronchi, and partly lies in their interstices between the fibres of elastic tissue by which the air-cells are entwined. The hemorrhage does not produce laceration of the lung-substance. The abrupt boundary of a hemorrhagic infarction is caused by the fact that the bleeding only comes from the capillaries belonging to a single twig of the pulmonary artery. The range of the capillary system of an artery depends upon its size; hence hemorrhagic infarctions which arise within the capillary limit of a large branch of the pulmonary artery are far more extensive than one which forms about a smaller twig. As the main trunks of the pulmonary artery enter the roots of the lungs in company with the great bronchi, and ramify toward the surface, constantly growing smaller by repeated subdivision, until each ultimate twig terminates in a single lobule, the reason is plain why the infarctions occurring in the interior of the lung are large, and why the peripheral infarctions preserve both the size and the cuneiform shape of the superficial lobuli. Upon careful examination of an arterial branch, within whose range a hemorrhagic infarction has formed, we find in it a clot, by which its calibre is more or less obstructed. This is easily demonstrated in the larger branches, but in the very small ones it is sometimes quite difficult. (*Niemeyer.*)

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[1] In another case of this sort, which will be related in Chapter XI., (see Case LXI.,) the pulmonary extravasation was very extensive, and assisted materially in producing the fatal issue.

That the obstructing clot is not formed at the place of lodgment, but comes from some remote part of the body whence, on getting detached, it was swept along in the current of the blood, until, finally, it became impacted in some branch of the pulmonary artery too narrow to admit of its passage, has long been recognized as the condition under which hemorrhagic infarction arises. The credit of this valuable discovery is due to Virchow. That investigator, after introducing particles of fibrin, muscle, elder-pith, etc., into the jugular veins of dogs, demonstrated by dissection that these foreign bodies plugged up branches of the pulmonary artery, and produced hemorrhagic infarctions, lobular pneumonia, and small abscesses, beyond the obstructed points. Conversely, he proved by dissection of bodies, in which the diseased spots long known as pulmonary metastases were found, that each artery leading to an affected point was occluded by an embolus or fibrinous plug, which, in all probability, had come from some peripheral vein that was thrombosed, or from particles whose origin could be traced to some region of suppurative or sanious ulceration upon the surface. (*Niemeyer.*)

It is easy to understand how hemorrhagic infarctions of the lungs are produced by embolia that have originated in disintegrating thromboses of peripheral veins. When embolia have become detached from their place of origin by the current of the blood, they meet with no obstacle on their way to the heart, for the veins through which they travel are constantly growing larger. They pass unhindered into the right heart and into the pulmonary artery, and do not get arrested nor impacted until they arrive at such branches of the latter as are too small to admit them. (*Niemeyer.*)

The hemorrhagic infarctions of the lungs which so often occur in cases where the heart is diseased, and especially in cases where the mitral value is insufficient, are also due to embolism of the pulmonary artery, as has been shown by Rokitansky and Gerhardt. The embolia which plug up the branches of that artery in cases of heart-disease do not come from the peripheral circulation, like the embolia which produce the so-called metastatic infarction, but from the right side of the heart itself, and especially from the right auricle; for coagula that are firmly entangled in the trabeculæ are apt to be formed in this chamber, as a consequence of venous stagnation, in all cases where there are mitral insufficiency and mitral regurgitation of a well-marked character. Now if, in one of these cases, a particle of coagulum happens to be broken off and washed away by the current of the blood, some branch of the pulmonary artery becomes obstructed by it, and hemorrhagic infarction of the corresponding lung ensues. The embolia which are thus derived from cardiac thromboses are generally larger than those which come from the peripheral veins. This circumstance readily explains why the infarctions of heart-disease are generally more extensive than metastatic infarctions, and likewise why the former are often found in the interior of the lung, near its root, while the latter are generally situated near the periphery. As very minute embolia also may be washed away in cases of cardiac thrombosis, we likewise see how, besides the larger infarctions at the roots, smaller peripheral ones also occur in heart-disease. (*Niemeyer.*)

We have next to explain the process by which capillary hemorrhage is induced in these cases of pulmonary embolism, a process which, at the first glance, seems by no means easy of elucidation. The theory of Rokitansky, "that occlusion of the minutest arterial branches of the lung and its capillaries causes a collateral hyperæmia, which results in hemorrhage and exudation," is unsatisfactory, for, in such cases, the extravasation does not issue from the neighboring capillaries, but from those of the obstructed vessel itself. Virchow, also, does not give a satisfactory explanation of this form of capillary hemorrhage. It is, therefore, all the more welcome and interesting that Ludwig has furnished a complete and final solution of the problem, from an entirely unbiased point of view, by showing the influence which the obstruction or incomplete occlusion of an artery, by an embolus, exerts upon its capillaries. His words are as follows: "Tension within the artery below the place of constriction is diminished, since a liquid flowing through a narrow tube loses more of its impetus than in flowing through a wide one. But we must not infer from this that, when an artery is constricted, the contents of its capillaries are lessened, and that the parts which they traverse grow paler. The sluggishness of the stream thus produced in the capillaries rather has the effect of allowing *the heavy blood-corpuscles to collect and become crowded together*; now, as two or more blood-corpuscles, if brought into contact, are apt to become permanently adherent, the blood itself can form a plug capable of closing the capillaries. Such an occurrence, which converts the capillaries into blind appendices to the artery, must cause an increase in its internal pressure." Let us add that, in consequence of this pressure, which, after the stoppage, is as great in the capillaries with thin, delicate walls, as in the afferent vessels themselves, the distended capillaries burst, and extravasation of blood ensues. This furnishes a simple and entirely satisfactory explanation of the process by which capillary hemorrhage is induced in cases of pulmonary embolism, and gives the reason why it is limited to the region supplied by the occluded artery. The utmost that can be said against the accuracy of this explanation is, that the artery leading to a pulmonary infarction not only is narrowed, but is entirely closed. Such an objection, however, is untenable. The lodgment of an embolus, which, we find, usually takes place at the bifurcation of an arteriole, very rarely produces absolute closure of the vessel at first, but merely occasions in it more or less obstruction. Afterward, when the infarction has become established —an event requiring but little time—fibrin is deposited upon the embolus, and the closure of the vessel becomes complete. (*Niemeyer.*)

Anatomical Appearances.—The hemorrhagic infarctions of the lungs which are produced by disease of the heart, vary in size from a hazel-nut to a hen's egg. They are of a blackish-red or blackish color, completely inelastic, and void of air, so that they feel from without like hard knots. Their cut surface presents an irregular, coarse, granulated aspect, from which a brownish-black mass may be scraped off with the scalpel. In the immediate vicinity of these sharply-defined spots the pulmonary tissue is usually full of blood, and œdematous, from collateral fluxion. Their seat, as already mentioned, is usually at the middle of the lower lobes, or near the roots of the lungs; occasionally at the surface. Microscopic examination shows that the affected capillaries are distended with blood-corpuscles, and that collections of them are also formed in the pulmonary tissue outside of the capillaries. (*Niemeyer.*)

When the infarction is of long standing, it looks paler and yellowish, the fibrin having undergone fatty transformation, and the coloring-matter of the blood having become partially decomposed. Still later, the fatty fibrin is absorbed, part of the hæmatin is turned into pigment, and the only remaining trace of the infarction is a blackish induration of the pulmonary substance. In the rare cases wherein an abscess forms, it may become incapsulated, and its contents may thicken into a cheesy or calcareous mass. (*Niemeyer.*)

The hemorrhagic infarctions of the lungs which are produced by embolia derived from the peripheral veins have generally a small size, (varying from that of a pea to a cherry,) a cuneiform shape, and a superficial situation, as already mentioned. These so-called metastatic infarctions are entirely similar to those which arise from heart-disease, in color, consistence, and friability. The microscope also reveals the same appearances. (*Niemeyer.*)

When metastatic infarctions terminate in metastatic pneumonia or abscesses, the process of discoloration and disintegration usually commences in the centre of each diseased spot; cavities form, filled with a yellowish mass, which consists of *débris* of the pulmonary substance, and of products resulting from the molecular decay of the extravasated blood and fibrin, but which at first does not contain any purulent matter, nor any pus-corpuscles. Upon pouring water over the cut surface, we can see the vestiges of the lung-tissue floating in the hollows. The process of disintegration gradually spreads, until scarcely a trace is left of former thickening, even at the periphery of the abscesses. When they are situated immediately under the pleura, yellowish deposits of false membrane form upon the latter, and beneath it lie the infarctions, each "forming a rounded-nodular prominence like a furuncle." (*Rokitansky.*)

Pulmonary gangrene also is occasionally produced by hemorrhagic infarction of the lungs. To discuss this topic, however, is foreign to our purpose.

Symptoms and Course.—Hemorrhagic infarction of the lungs, when produced by chronic disease of the heart, is oftentimes attended with such well-marked and unequivocal symptoms, that its presence can be recognized with perfect certainty. In other cases, however, the diagnosis is difficult or quite impossible.

The symptoms from which we can infer that one or more pulmonary hemorrhagic infarctions have been formed in cases of heart-disease are the sudden occurrence of dyspnœa, which may be so great as to threaten suffocation and cough, with the simultaneous appearance of a peculiar sputum tinged with blood. In some instances the physical signs of a circumscribed condensation of the pulmonary tissue are also present. It is obvious that the occlusion of one or more branches of the pulmonary artery with embolia may produce extreme dyspnœa. In order for the process of breathing to be normally conducted, it is necessary that the air in the pulmonary vesicles and the blood in the pulmonary capillaries should both be properly renewed, and, therefore, when the access of blood to, or the entrance of air into, any considerable part of the lung is prevented by the obstruction of a bronchus, or by the plugging of an arterial branch, the effect upon the respiration is equally embarrassing. The sputum of pulmonary hemorrhagic infarction bears a considerable resemblance to that of pneumonia, but it is generally less tough in consistence, and is darker in color; moreover, the expectoration of this sputum usually continues much longer than the expectoration of the pneumonic sputum. The former may persist for a week, or even a fortnight. Circumscribed condensation of the pulmonary tissue cannot be detected, unless the hemorrhagic infarction is of comparatively large size, and has extended to the surface of the lung. The percussion-sound then becomes dull, and crepitation with bronchial sounds is audible over a limited portion of the chest. Such cases, however, are rare. The diagnosis of pulmonary hemorrhagic infarction may be confirmed, a few days after the attack of dyspnœa with bloody expectoration, by the development of extensive pneumonic infiltration, or by the occurrence of inflammatory effusion into the pleural sac, for hemorrhagic infarctions often produce extensive inflammation of the surrounding pulmonary tissue, and still more frequently cause inflammation of the pleura.

The symptoms above described are all immediately dependent upon the plugging up of one or more branches of the artery. Now, in many cases, we find other symptoms, which result from thrombosis of the right heart, and hence are to be regarded as indirect tokens of hemorrhagic infarction. These are, a sudden irregularity of the pulse, a sudden increase in the area of cardiac dulness, and the sudden cessation of an adventitious cardiac murmur that had previously existed. This sudden subsidence of a loud morbid heart-sound is not only a most striking occurrence, but it is generally very significant, and of great diagnostic importance. And when this group of symptoms coexists with that which is above described, the picture of pulmonary hemorrhagic infarction becomes complete. But the embolia, broken off and washed away from cardiac thromboses, may be so small that they do not produce any characteristic phenomena; therefore, we may still confidently diagnosticate pulmonary hemorrhagic infarction whenever unequivocal signs of disorder in the pulmonic circulation and of capillary hemorrhage from the lungs suddenly occur in the course of chronic heart-disease, although the pulse continues regular, the area of cardiac dulness remains unchanged, and no signs of cardiac thrombosis are present.

Finally, if we bear in mind that the characteristic expectoration of blood from the air-vesicles is not always observed in hemorrhagic infarction, and besides, that violent fits of dyspnœa may arise from a great variety of causes in disease of the heart, and that infarctions, seated deep within the lung, cause no physical signs, it is easy to understand why the disease, which in many cases does not present the smallest difficulty of diagnosis, may sometimes elude detection and even suspicion—as, for instance, where the patient is already extremely short of breath and dropsical, or is otherwise wretchedly ill. In the dissection of cases of diseased heart, therefore, we should be prepared to find hemorrhagic infarctions as "accidental discoveries," where their existence has not been suspected. (*Niemeyer.*)

When hemorrhagic infarction of the lungs is produced by embolia which have come from the peripheral veins, that is, when the so-called metastatic infarction of the lungs occurs, the symptoms usually differ considerably from those which are described above. The fluid products of an unhealthy inflammation or ulceration almost always pass into the circulation along with the embolia; and while the latter give rise to metastatic infarctions, the former induce the symptoms of pyæmia or septicæmia, according to the nature of the case; for example, intense fever, violent rigors, great debility, purulent inflammation of serous membranes, etc. Hence we find that most patients who have metastatic infarction of the lungs are in a state of extreme prostration, that their sensorium is blunted by the poisoned state of their blood and by the intensity of the asthenic fever, and that they do not complain of difficult breathing nor pain in the side or breast, nor show any disposition to cough. The intense dyspnœa which occurs in cases where large branches of the pulmonary artery are obstructed with embolia, does not exist in cases of metastatic infarction, for the occluded vessels are almost always very small; and dyspnœa of a slighter degree is not noticed by the patient, because of his stupefied condition. The characteristic sputum also is almost always absent, for generally the patient neither coughs nor expectorates. Finally, notwithstanding their superficial position, metastatic infarctions scarcely ever occasion circumscribed dulness on percussion, or bronchial breathing in the affected part. But in most cases where metastatic infarction of the lungs occurs, the symptoms which might otherwise be developed are masked by the hebetude that results from the pyæmia or septicæmia with which it is accompanied.

Treatment of Pulmonary Hemorrhagic Infarction.—From the nature of the disease itself the treatment must be principally directed to the relief of prominent symptoms. When it results from disease of the heart, we must be careful not to attribute the sudden occurrence of dyspnœa to sudden aggravation of pulmonary hyperæmia, for, as shown above, the real cause of dyspnœa in these cases is anæmia of considerable portions of the pulmonary tissue. Venesection employed in such a case might increase a collapse of the lung already present, and might hasten a fatal issue. It is only when the infarctions have induced collateral hyperæmia and collateral œdema in the rest of the lung, and when the dyspnœa is plainly due in great measure to this cause, that cautious blood-letting, either by cupping or venesection, is ever admissible. As a general rule, until the pulse, which usually is feeble, grows stronger, and until the skin, which usually is cool, becomes warmer, we must confine our treatment to the administration of stimulants, and to the application of sinapisms and jugs of hot water to the extremities. The hæmoptysis is almost never so abundant as to call for the exhibition of any hæmostatic remedies. The inflammation of the pulmonary tissue or pleura, which not unfrequently sets in at a later period, may demand local depletion and the application of poultices, with other antiphlogistic measures. (*Niemeyer.*)

We shall now proceed to briefly consider the *second variety of pulmonary extravasation*, or so-called apoplexy of the lungs, namely, that in which the blood is effused from vessels of some considerable size, the pulmonary tissue is more or less torn or broken down by the extravasation itself, and cavities filled with blood, of corresponding size, are formed in the pulmonary parenchyma. As remarked by Trousseau, and already mentioned by us, pulmonary extravasation has occasionally proved fatal in a sudden manner and been mistaken for cerebral apoplexy. On examination post-mortem, the brain and its membranes have been found to be normal, but extravasated blood has been discovered in the pulmonary parenchyma. Some cases of this sort have been recorded by Corvisart, Bayle, and Andral, wherein death occurred almost instantaneously. It is but very seldom, however, that pulmonary extravasation proves fatal in so sudden a manner.

Etiology.—In this form of pulmonary hemorrhage the pulmonary tissue is destroyed by extravasated blood, and an abnormal cavity is established. Capillary hemorrhage scarcely ever breaks down the pulmonary tissue. It is only erosion or laceration of the larger vessels, especially rupture of the arteries, which produces destruction of this kind. In rare cases atheromatous degeneration of the pulmonary artery causes aneurismal dilatation, and finally the rupture of it; but, generally, wounds, contusions, or concussions of the thorax are the causes which produce this form of pulmonary extravasation. (*Niemeyer.*)

Anatomical Appearances.—A cavity is found in the lung, containing both liquid and coagulated blood, and surrounded by shreds of the lacerated pulmonary substance. If the extravasation has its seat in the periphery of the lung, the pleura, too, is often torn, and blood is effused into its sac. Such hemorrhages are almost always fatal, and therefore we have but little knowledge of the mode of repair which nature adopts in such cases. (*Niemeyer.*)

Symptoms.—Violent and rapidly-fatal hæmoptysis, following serious injury of the thorax, or, in other cases, suffocation from effusion of blood into the bronchia faster than it can be expectorated, or sudden death from internal hemorrhage, may be the symptoms of this exceedingly rare disease, which, being absolutely deadly, is susceptible of no treatment. (*Niemeyer.*)

Thus we have shown, in the foregoing pages of this chapter, that pulmonary extravasation may be due either to a diseased or hemorrhagic state of the blood itself, on the one hand, or to some lesion of the pulmonary blood-vessels, such, for example, as rupture of their coats, embolism of their calibre, etc., on the other. But, at the same time, there is reason to believe that pulmonary extravasation may also be occasioned by some injuries and diseases of the brain, through the powerful influence which they exert upon the sympathetic nervous system. We infer that extravasation of blood into the pulmonary parenchyma may be produced in this way: firstly, because we sometimes meet with such extravasations in the lungs of well people who have died in a few hours from injury of the brain, there being neither blood-disease nor embolism in their cases; and, secondly, because of the very interesting results of a similar character that have been obtained by experimenting on animals. Dr. Brown-Séquard has recently published the following statements, which afford a fit illustratien of the last-named point. He says: "In making experiments on the comparative lethality of injuries to the left and the right side of the brain, I found, a year ago, that one of the most frequent causes of death, when it does not occur immediately or very soon after wounds of the brain, in guinea-pigs especially, was pneumonia. I was led by this fact to perform a large number of experiments, to ascertain the immediate effects of an injury to the brain on the lungs. The results obtained were startling; in almost all cases of injuries by crushing or section of the pons Varolii ecchymoses were found in the lungs. Sometimes the whole lung was crowded with effused blood, and real pulmonary apoplexy [or extravasation] existed. In some instances the effusion took place in the bronchial tubes. Injuries to other parts of the base of the brain, especially the crura cerebri and crura cerebelli, sometimes are followed by the same effects on the lungs; and it is extremely probable that a slight pressure upon the pons Varolii by effused blood is sufficient to produce it. Injuries to the medulla oblongata and to the spinal cord have but very rarely (only in three or four experiments out of a great many) caused an effusion of blood in the lungs. This is the more remarkable because, without any doubt, the nerve-fibres going from the pons Varolii to the lung, which cause the rupture of the small blood-vessels in this viscus, pass through the medulla oblongata and the cervical part of the spinal cord. Many experiments have shown me that it is not through the par vagum, but through the sympathetic nerve, especially by its spinal roots, which throw themselves in the first thoracic ganglion, that the peculiar influence of the irritated pons Varolii exerts itself in producing a pulmonary hemorrhage, [or extravasation.]

"A hemorrhage is not the only immediate effect that can be observed after an irritation of the base of the brain by crushing or cutting; an anæmic condition, œdema, and emphysema can also be produced. Some small parts of the lungs are found perfectly white, and, according to the examination of a distinguished microscopist, M. Ranvier, who has kindly helped me in some of these researches, absolutely deprived of blood, no doubt through a spasm of the blood-vessels having emptied them of their contents. This may occur after injuries of almost all parts of the base of the brain, but especially the pons Varolii. Not so as regards œdema, which principally appears after an injury to the medulla oblongata. Looking at a lung in which such an alteration exists, one observes one or several grayish spots, generally circular, and of the size of the head of a pin, protruding as the part of a sphere from the surface of the lung. This pearl-like part of the lung, according to my able friend M. Ranvier, contains a good deal of serum, and its minute blood-vessels are filled with the white corpuscles of blood, and free from red corpuscles. This is, indeed, a most wonderful fact, and the more so that this change in the contents of the pulmonary capillaries is immediate.

"The last effect I intend to mention of an injury to the base of the brain on the lung is already known to experimenters: I mean emphysema. But what I will state about it is a new and very remarkable fact, which does not agree with the reigning theories of the production of emphysema. It is that this morbid condition can appear when not a single respiratory movement takes place, after an irritation of the base of the brain either by crushing or cutting.

"When I publish the details of my experiments on the influence of injuries to the brain on the lungs, I will show that, in man, diseases of or injuries to the brain very frequently produce organic alterations in the lungs. I will content myself here, to prove the frequency of that morbid influence of the brain on the pulmonary organs, to state that, out of one hundred and eighty-eight cases of organic diseases of the brain recorded by Calmeil, there was a morbid state of the lungs, especially inflammation, in more than sixty cases, that is, in one case out of three. I have no doubt that many patients attacked with brain-diseases die from a disease of the lungs caused by that of the central organ of the nervous system." (Vide *American Journal of the Medical Sciences*, for April, 1871, pp. 561, 562; also the *Lancet* for January 7th, 1871.)

From what has just been said we infer, then, with good reason as we think, that pulmonary extravasation and other pulmonary lesions of importance may be produced, in man, by diseases and injuries which involve the base of the brain. In Chapter XI. two cases of epidemic cerebro-spinal meningitis will be related which support this view. (See Cases LVIII. and LX.) In both of them the cerebral phenomena were strongly marked. In one of them, pulmonary extravasation, and in the other, pulmonary carnification, was found at the autopsy. This subject is of great importance, and we therefore commend it to the careful attention of clinical observers and experimental pathologists.

In 580 cases of sudden death, Ogston found pulmonary extravasation in 36 instances which he groups together and presents in tabular form. (Vide *Ogston on Sudden Death, in British and Foreign Med.-Chirurg. Review*, Table iv. vol. xliv. 1869, pp. 458, 459.) In *four* of these the cause of death was fracture of the skull, in *one* cerebral apoplexy, in *one* injuries of the chest, in *one* stabbing, in *one* hæmoptysis, in *two* pneumonia, in *one* fatty heart, in *one* thrombosis of the right heart and pulmonary artery, in *one* peritonitis, and in *twenty-three* the pulmonary extravasation itself. Thrombosis of the right heart, however, was present in all in *five* instances, and of both sides of that organ in one instance, but Ogston considers this lesion to have been the cause of death in only one instance. Furthermore, of these 36 cases of pulmonary extravasation, 27, or three fourths, were males, and 9, or only one fourth, were females. (Vide *ibid.* p. 475.)

Concerning the occurrence of pulmonary extravasation Ogston makes the following important remarks: "The way in which pulmonary apoplexy presents itself in many cases is a puzzle to the pathologist, and it—more perhaps than any other morbid alteration—is found complicating cases where there already exists an evident and sufficient cause of death." . . . . "To take only one instance of this from the table; there are there noted four cases of fracture of the skull in which pulmonary apoplexy was present, and in each of them its extent varied; in one, it existed merely as two apoplectic depots in one lung; in another, the whole of the lower lobe of the left lung was consolidated from this cause; in a third, the back part of both lungs was affected; while in the fourth, the total extent of both lungs was the seat of the disease, there not existing a single portion of either lung, however small, which was not black, consolidated, and nearly airless, from infiltration of blood. This last case, occurring as it did in a man whose assailant was afterward tried for culpable homicide, left the medical man engaged in the case in an unenviable fix. The injuries in the head were confined to fracture of the basis cranii, to injury of the brain, and to effusion of blood among the membranes of the brain, but not enough in quantity to have caused the slightest compression. The man was known to have been in perfect health the moment before receiving the injury. I saw him dead half an hour later, and yet here were two causes of death, each in itself sufficient, and none of which could have existed before the injury was received.

"The explanation which suggested itself to me was the following, and it may be applicable, to a certain extent, to more of the cases in the table than those where fracture of the skull caused death. In the case just cited the natural mechanism of death after the injury of the brain would be by coma, and hence collection of blood in the right side of the heart and in the lungs, causing congestion of these organs. Now, pulmonary apoplexy is merely an advanced stage of congestion, where the blood is infiltrated into the substance of the lung-tissue instead of being retained within the vessels. What was the cause which changed congestion into extravasation is not clear; it may have been excessive heart's action, or some local cause seated in the lungs; but even this imperfect explanation removes the difficulties of the case, by converting the pulmonary apoplexy from a primary to a secondary cause of death." (Vide *Ogston on Sudden Death, in British and Foreign Med.-Chirurg. Review*, vol. xliv. 1869, pp. 474, 475.) Dr. Brown-Séquard's researches and experiments, however, afford a clear and satisfactory explanation of the difficulties mentioned above. Moreover, Dr. Ogston's cases support Brown-Séquard's views.

The so-called *apoplexy of the liver*, *spleen*, *kidneys*, etc., like pulmonary apoplexy, consists in the extravasation of blood into the substance of these organs. But such extravasations are generally not attended with any of the symptoms which the term apoplexy denotes. Hence, the employment of the word apoplexy to designate these parenchymatous hemorrhages is, to say the least, inappropriate, and should be abandoned. They can, however, be designated as hepatic, splenic, or renal extravasations, according to the organ affected by the hemorrhage, with perfect propriety. Again, hepatic, and splenic, and renal extravasations may consist either of hemorrhagic infiltrations derived from the capillaries or of hemorrhagic collections derived from the larger vessels that have been ruptured. Furthermore, capillary hemorrhage or extravasation as met with in the substance of the liver, spleen, kidneys, etc., is not unfrequently due to circumscribed *embolism* of these organs, and such cases should be designated as instances wherein *hemorrhagic infarction* of these organs has occurred. For an excellent illustration of the phenomena which attend hemorrhagic infarction of the kidneys and spleen, see Case XXXIII., in the Chapter on Cerebral Embolism, (No. VI.)

CHAPTER X.

ON THE SYMPTOMS, DIAGNOSIS, PROGNOSIS, AND TREATMENT OF APOPLEXY.

1. *Symptoms of Apoplexy.*—The premonitory symptoms described; some that are considered peculiarly ominous; but apoplexy often occurs without warning.—The signs of general plethora stated.—Cerebral hyperæmia often attended with depression of the cerebral functions; symptoms of depression affecting the sensibility; motor symptoms of depression, and psychical symptoms of depression enumerated; state of the pupils; state of the pulse and respiration; vomiting.—Cerebral hyperæmia not unfrequently attended with disturbances of the sensibility of an irritating character; they are stated.—In a third form of cerebral hyperæmia the mental symptoms predominate; they are described.—The symptoms which attend the fits of apoplexy are next given in full.—2. *Diagnosis of Apoplexy.*—When the attack occurs in the presence of witnesses the diagnosis is generally not difficult.—The following pathological states may, at first sight, be mistaken for apoplexy: 1. A state of coma when produced by alcoholic intoxication. 2. A comatose condition when produced by poisoning with opium or morphia. 3. Coma occurring in consequence of injury of the head, such as concussion of the brain, depressed fracture of the skull. 4. Coma when induced by epilepsy or eclampsia, the convulsive movements having ceased before the patient is found, but the symptoms of coma being unabated. 5. Coma the result of uræmia occurring as a consequence of renal disease. 6. Syncope with complete loss of consciousness, the result of fatty degeneration of the heart. 7. Syncope when produced by the direct action of the sun's rays, and therefore occurring as one of the leading varieties of sun-stroke.—How to distinguish apoplexy from each of these affections is pointed out.—Concerning throbbing of the carotids as a symptom of apoplexy; its proximate cause and what it indicates pointed out.—3. *Prognosis of Apoplexy.*—The prognosis of this disease as stated by Hippocrates and Celsus.—Some bad prognostics mentioned.—But the recovery sometimes is rapid and complete in very bad cases of congestive apoplexy.—A case in point related.—4. *Treatment of Apoplexy.*—1. On the remedial measures to be employed before the attack, that is, the prophylactic or preventive treatment of apoplexy. It is at this time that the physician can do most good to those who are liable to have apoplexy.—In no other disease are the causal indications of more importance.—The preventive treatment of apoplexy is minutely described.—2. *On the treatment to be employed during the fit of apoplexy.*—After the stroke has occurred the physician is comparatively powerless to benefit his patient.—Then he is practically limited to combating the more dangerous symptoms, or, in other words, to obviating the tendencies to death.—Concerning the employment of venesection and purgatives.—Venesection but seldom advisable, and the indications for its use pointed out.—Concerning the employment of topical bleeding.—The administration of purgatives generally required.—Emetics have been highly recommended; the indications for their employment mentioned.—Concerning the use of repellents and revulsives; cold affusion of the head; the application of the ice-bag or of frozen compresses to the head.—Celsus's remarks.—Dr. West's observations.—The author's experi-

ence on this point.—Concerning the use of revulsives or derivatives.—The administration of aconite.—The treatment of *nervous apoplexy* laid down.—The treatment of *infantile* apoplexy discussed.—3. *On the treatment to be employed after the attack of apoplexy.*—The man who has had one fit of apoplexy is strongly predisposed to have another, which will use him still more severely.—What he should avoid is stated.—Concerning the employment of counter-irritants and sedatives in such cases.—They are sometimes very useful.—Blistering was recommended by Sydenham.

1. Symptoms of Apoplexy.—This disease never occurs in people who are really well, although patients not unfrequently seem to be in the full bloom of health when attacked by it. In such cases, however, the robustness is only apparent; for, at the same time, there is also present a disordered state of the brain itself, or of the cerebral blood-vessels, or of the circulating blood, or of the heart, which is the forerunner of the apoplectic seizure.[1] The symptoms which are produced by these precursory disorders of the head or heart are, in general, the premonitory symptoms of apoplexy. In some cases, these premonitory symptoms are developed so gradually, or are so slight, as to attract but little or no attention, while in others they are so strong as to constitute veritable warnings. The symptoms which indicate that an attack of apoplexy is about to take place may be briefly summed up as follows: The sudden occurrence of headache, with a sense of heaviness and fulness in the head, and dizziness or vertigo, unusual noises or an unusual sense of throbbing or pulsation in the ears, muscæ volitantes, flashes before the eyes, general nervous irritability or hyperæsthesia, disturbed sleep, a flushed and turgid appearance of the face and eyes, with a corresponding increase of the cephalic temperature, or an appearance of general plethora, a heavy or stupid look, absent-mindedness, forgetfulness or loss of memory, sudden impairment of sight, with dulness, drowsiness, numbness and formications in the extremities, incoherency of ideas, and indistinctness of speech. Some symptoms are considered to be peculiarly ominous; for example, transient or evanescent feelings of formication and numbness in certain limbs, momentary loss of memory for some words and figures, and temporary paralysis confined to certain groups of muscles. These premonitory symptoms or warnings of apoplexy are, in some cases, due to cerebral hyperæmia; in others, to partial or circumscribed anæmia of the brain resulting from degeneration of the walls of its blood-vessels. But as already intimated, apoplexy

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[1] We have elsewhere shown that cerebral hyperæmia does not induce the symptoms of apoplexy in subjects where the cerebral substance, the cerebral blood-vessels, and the blood itself are in a healthy state; that apoplectiform cerebral hemorrhage does not occur unless the walls of some of the cerebral arteries have become much weakened by disease; that apoplectiform cerebral embolism does not take place unless the left chambers of the heart or some other distant organs are affected by disease in such a way as to furnish embolia which, when washed off by the blood-stream, may be carried to the cerebral arteries and plug them up.

not unfrequently occurs without the presence of any symptoms that are recognized as warning.[1]

General plethora is so frequently concerned in the causation of apoplexy that the signs which denote its presence should be mentioned in connection with the premonitory signs of apoplexy. In such cases the face appears full and turgid, with a purplish tinge. The eyes seem rather small, and the conjunctivæ more moist than usual. There is distention of the capillaries, as can be observed on the lips and mucous surfaces. The pulse is large, somewhat hard, and resistant. There is also a turgid appearance of the veins. Obesity is sometimes an accompaniment, though by no means an infallible sign, of plethora. Indeed, many fat persons suffer from a deficiency rather than from an excess of blood. (*Tanner.*) Plethora also produces lassitude and indolence; a desire for sleep, which is often accompanied with snoring and dreaming; a liability to fits of vertigo and headache; and sometimes attacks of hemorrhage, especially from the nose or from congested piles. (*Tanner.*)

Cerebral hyperæmia also plays so important a part in the production of apoplexy that some of its symptoms deserve a more attentive consideration at our hands than they have yet received. Oftentimes cerebral hyperæmia is attended with depression of the cerebral functions. With respect to the *sensibility*, the symptoms of depression which have been observed are a remarkable insensitiveness, a tolerance of external irritation, bright light, loud noise, etc. The patient takes no notice of slight irritation. When this state of incomplete cerebral anæsthesia is increased to complete cerebral anæsthesia, the patient does not perceive even the strongest irritation. The excitability of the parts of the brain through which external impressions are perceived is lost. The *motor* symptoms of depression are decided slowness or sluggishness in the movements of the patient: "his limbs are as heavy as lead." If this state increases he loses all power of making voluntary movements. The excitability of the motor centres is extinguished, and cerebral paralysis occurs. The *psychical* symptoms of depression are, loss of interest, and indifference; great slowness of thought, and limitation of the ideas; inclination to sleep, from which the patient is roused with difficulty, and subsequently cannot be roused at all. When this state is increased to the highest point, the consciousness is completely lost. (*Niemeyer.*)

Again, in hyperæmia of the brain, abnormities are not unfrequently observed in the movements which are excited by the cerebral nerves, independently of the will. For example, in states of irritation the *pupil* is contracted, because the oculo-motor nerve is more excited; in depression it is dilated, because then the sympathetic filaments of the iris act more strongly. In just the same way as observed in physiological experiments, irritation of the vagus causes the heart to beat slower, while in central paralysis of the vagus, as well as after its division, the heart's action becomes more frequent. In paralytic states of the brain the respiration is often very slow, deep, and stertorous. Although we cannot give an exact explanation of this symptom, we may still call attention to the fact that, after division of the vagus in animals, besides other symptoms, there is always retardation of the respiration. Lastly, we must mention *vomiting* as a very frequent and important symptom of cerebral irritation. Without understanding the process in the brain by which the act of vomiting is brought about, we know that it is artificially excited in animals by dividing the vagus, and then irritating the cerebral end. (*Niemeyer.*)

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[1] **The warning symptoms of apoplexy bear a strong resemblance to those of cerebral hemorrhage, of which we have endeavored to give a very full account in Chapter V.; and that part of said chapter may be consulted with advantage in this connection.**

In many cases of cerebral hyperæmia, disturbances of sensibility, of an irritating character, are the most prominent symptoms. Such patients complain of the head feeling contracted, and of more or less severe headache; they are sensitive to bright light and loud noises; have flashes of light before the eyes, and noises in the ears. They go to sleep with difficulty, and the sleep is disturbed by unquiet dreams. In severer cases, dizziness and a sense of formication in the extremities are often present. The face and conjunctivæ are usually reddened, the pulse full and rapid. But we must not consider these symptoms as constant; for, in the most dangerous cases of cerebral hyperæmia, in those induced by excessive mental labor, continued night-watching, etc., the amount of blood in the external organs does not correspond at all to the supply in the brain; and frequently in such patients the conjunctivæ are not injected, and the face is not flushed, but, on the contrary, they are quite pale. (*Niemeyer.*)

In a third form of cerebral hyperæmia, the mental symptoms predominate to such a degree that the disease is often mistaken, and, to the great injury of the patient, is sometimes considered as an attack of melancholy; at others, as mania. In the former case, after a few days of headache, disturbance of sensibility, and sleeplessness, the patients are seized with an undefined feeling of anxiety and disquiet. They cannot stay long in one place, go about restlessly, are worried, and are conscience-stricken about slight oversights. There is also delirium, which has the same character as the above-described frame of mind, and results from the attempts to explain it. At first the patients struggle against this delirium, which they occasionally recognize as such, and which they fear, as they think they are "out of their minds;" but they soon get weary of this struggle, and give it up. In such cases the sleeplessness is almost absolute; opium, given by ignorant physicians, has no effect, or, after the exhibition of this remedy, which is injurious and dangerous to the patient, there is a short restless sleep, from which the patient awakes with all the symptoms increased. In this form of cerebral hyperæmia, which chiefly occurs as a result of excessive mental labor, there is usually a frequent pulse and other symptoms of fever; but in these very cases the increase in the quantity of blood contained in the brain is not attended with a corresponding increase in the amount of blood in the face; such patients are not high colored, but often they are even pale. In consequence of the fever and sleeplessness they rapidly lose strength, emaciate, and, if they do not fall into the right hands, they are in great danger of dying from their disease. Finally, the excitement gives way to apathy, the insomnia to deep sleep, from which the patients cannot be roused, and in which they die. Far more rarely, there is permanent mental disease. (*Niemeyer.*)

In other persons, where there is also excessive psychical disturbance, it appears in the form of maniacal attacks, with corresponding delirium. This form is especially seen after a long-continued excessive use of spirituous liquors, in that class of topers who for a year will use very little liquor, but when they have begun to drink do not know when to stop. This cannot be easily mistaken for an attack of delirium tremens. The patients are sleepless, run about, fight and bite if they are held, destroy any thing that comes in their way, cry, laugh, or sing. There is also delirium of varying character; usually the patients consider themselves as injured and betrayed, and rage against their enemies and persecutors. The continued muscular exertion throws them into a perspiration, the heart-beat and pulse are accelerated and stronger, and the face is usually reddened. This form of cerebral hyperæmia is also very dangerous, if its nature be mistaken and it be improperly treated, for then an apoplectic attack, a true apoplexy, or an excessive hyperæmia of the lung with acute œdema, causes death. According to my own observations, says Niemeyer, cases exactly like the following, detailed by Andral, are by no means rare; in the Magdeburg hospital, and in the Greifswalder clinic, I have seen several of them within a few years: "For several days a middle-aged man kept up a series of cries sufficient to disturb the whole hospital-ward. These cries ceased suddenly, and on approaching his bed he was found dead. He would not have died more suddenly if struck by lightning. On opening the body the only lesion found was a lively injection of the cerebral substance." (Vide *Niemeyer's Text-Book of Practical Medicine*, vol. ii. p. 165, 1st Am. ed.)

Having described at sufficient length the phenomena which attend the principal varieties of cerebral hyperæmia that are liable to usher in attacks of apoplexy, we will now proceed to describe the symptoms which usually attend the apoplectic fits themselves. In some cases of congestive apoplexy the patient suddenly becomes dizzy; "every thing whirls around with him;"

he staggers, all looks dark before him; he loses consciousness and sinks to the ground, either with or without slight spasm. Such an attack, wherein all functional activity of the cerebrum is suddenly suspended, may speedily cause death by the paralysis extending from the cerebrum to the centres of the organic nervous system. Oftentimes, however, the patients recover consciousness after a while, but they do not retain any recollection of what has transpired during the fit.

In other cases, the stroke occurs entirely without warning. The victim is suddenly bereft of consciousness and precipitated into coma. If standing when attacked, he falls to earth as if stricken down by a powerful blow, and lies motionless just where he happens to fall; his respiration is generally slow, deep, and snoring; pulse full and slow; carotids throbbing; countenance dusky in hue and swollen in appearance; conjunctivæ injected; pupils contracted. But, not unfrequently, the face is pale instead of being purple, the pupils dilated, the pulse small, frequent, and irregular, and the respiratory movements obstructed by pulmonary œdema. Slight convulsive movements or twitchings are often observed in the muscles of the face and extremities.

If the patient is about to recover from the fit, his breathing becomes less deep and more natural, the stertor grows fainter and disappears, his face grows less swollen and congested, and, finally, his consciousness returns, he opens his eyes, and asks, "What is the matter?" In cases which terminate favorably, the total loss of consciousness may continue for a few minutes, half an hour, an hour, or several hours, and sometimes patients recover from apoplexy who have lain comatose more than twenty-four hours. The author has personal knowledge of one case of that description. Forbes Winslow mentions the following case in point: A lady was seized with a fit of apoplexy while playing whist. The attack occurred on Thursday evening; she remained unconscious until Sunday. Immediately on recovery she exclaimed, "What is trump?" (Vide *Forbes Winslow on the Obscure Diseases of the Brain*, etc. pp. 322, 323.)

If the patient is going to die, all his symptoms become worse or more threatening, the stertor grows louder, œdema of the lungs with rattles in the trachea appears, the pulse progressively becomes more frequent and irregular, the heart's impulse grows more feeble and its sounds more faint, the pupils dilate, the under jaw falls down, for the facial muscles have lost their contractility, the nose becomes pinched, the skin grows cold and bathed in clammy sweat, at last the respiratory movements cease, and death closes the scene.

In cases where cerebral hemorrhage sets in with apoplectic coma, which, as we have elsewhere shown, but seldom happens, comparatively speaking, it is usually attended with hemiplegia, and hence the patients belonging to this category generally, but not always, exhibit the symptoms of one-sided paralysis in addition to the phenomena of congestive apoplexy. In such cases, the mouth is observed to be drawn to one side, and the paralyzed cheek may be puffed out and drawn in again, with a loud noise, by the air, during the movements of expiration and inspiration.

Finally, in cases where the phenomena of apoplexy are induced by embolism of the cerebral arteries, the patient on regaining his consciousness not unfrequently says that, at the moment of attack, he felt exactly as if he were struck a violent blow in the head with some heavy substance. See Case XLII. in Chapter VI. for an illustration of this statement. In these cases also the attack is often attended with the sudden appearance of acute pain in the head, with screaming, etc.

2. Diagnosis of Apoplexy.—In cases where the attack occurs in the presence of witnesses, the diagnosis of this disease is generally not hard to make; for the symptoms of an apoplectic fit, in the ancient and clinical sense of the term, are so strongly marked as to impress themselves on the mind of even a very superficial observer. But, in cases where the attack does not occur in the presence of any body, as, for example, in cases where the patient is found lying in a state of insensibility which appears to be a state of apoplectic coma, after having been seen a short time previously in usual health, the establishment of the diagnosis on a satisfactory basis may give considerable trouble. Under these circumstances either of the following pathological states may, at first sight, be mistaken for apoplexy, for the symptoms which attend them not unfrequently bear considerable resemblance to those of apoplexy :—

1. A state of profound insensibility or coma when produced by alcoholic intoxication.

2. A comatose condition when produced by opium-poisoning, whether it be the result of accident or design.

3. Coma occurring in consequence of injury of the head, such, for example, as concussion of the brain, depressed fracture of the skull, linear fracture of the skull with rupture of the middle meningeal artery, etc.

4. Coma when induced by epilepsy or epileptiform convulsions, the convulsive movements themselves having ceased before the patient is found, but the symptoms of coma being unabated.

5. Coma the result of uræmia occurring as a consequence of renal disease.

6. Syncope with complete loss of consciousness the result of fatty degeneration of the heart.

7. Syncope suddenly produced by the direct action of the sun's rays, and therefore occurring as one of the leading varieties of sun-stroke.

The form of *coma* which is produced by *drinking spirituous liquor to great excess*, or *dead drunkenness*, as it is sometimes called, may generally be recognized by smelling the fumes of the liquor in the patient's breath, by finding an empty liquor-bottle in his pocket or somewhere else about him, and by other circumstances of a similar nature. In such cases, the characteristic odor of alcohol is usually appreciable; but implicit confidence should not be placed on this point, for a man who has been drinking may, at the same time, be attacked with apoplexy or cerebral hemorrhage. In drunkenness the urine is generally copious and limpid; in cases where a large quantity of spirit has been taken, the specific gravity of this secretion may even be found to be below that of water. According to Dr. Anstie, the presence of a poisonous dose of alcohol in the system can be determined, if the addition of one drop of the urine to fifteen minims of a chromic acid solution turns the latter at once to a bright emerald-green color. This chromic acid solution is made by dissolving one part of bichromate of potash in three hundred parts by weight of strong sulphuric acid. (*Tanner.*)

Nitro-benzole also has caused several deaths, in four or five hours, after giving rise to symptoms resembling those of drunkenness, followed by sudden coma. In such cases, the nature of the difficulty is easily recognized from the peculiar smell of the poison. (*Tanner.*[1])

Loss of consciousness and sensibility when due to *poisoning by opium* can often be correctly diagnosed from the highly-contracted state of the patient's pupils, and the great infrequency of the respiratory movements. The former may be contracted down to a mere point, and the latter be as low as only five or six per minute. It should not be forgotten, however, that as death approaches in cases of poisoning by opium, and the oculomotor nerve loses its irritability, the pupils not unfrequently cease to be contracted, (Vide Case XXIII.,) and sometimes become widely dilated. The remarkable slowness in the breathing, mentioned above, is a pretty constant and very valuable sign of opium-poisoning. Besides, the odor of laudanum is sometimes detected in the patient's breath, and an empty vial that has contained laudanum or morphia or some other preparation of opium is not unfrequently found on his person or in his room.

When a patient is found lying in a comatose condition in consequence of *injury of his head*, the nature of the case can, for the most part, be correctly determined by making a careful examination of the head itself, and by taking into account the circumstances attending the discovery of the body, together with its relation to surrounding objects. The examination of the head in such a case will generally reveal lacerations or bruises of the scalp, and, not unfrequently, a fracture of the skull with depression can also be felt.

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[1] So many cases of apoplexy occurring in the streets have been mistaken for alcoholic intoxication, that no person found insensible by the police (whatever the *supposed cause* may be) ought to be placed in a cell until a cautious examination has been made by a medical man. Even if the person be "dead drunk," treatment is sometimes urgently demanded in order to prevent a fatal termination. But if the case be one of apoplexy, putting aside the question of treatment, the feelings of the relatives surely deserve some consideration; for it must be no small aggravation to their grief to find that one whom they have respected and cherished has been locked up on a charge of drunkenness. (*Tanner.*)

To make the diagnosis between *epilepsy* and apoplexy is generally easy, (see Comments on Case XIII. ;) in some cases, however, as intimated above, it is very difficult, for these two disorders are closely allied to each other. A fit of epilepsy, with the convulsive movements left out, presents almost exactly the same symptoms as a fit of apoplexy. There is the same state of cerebral anæsthesia and paralysis of the mental functions, which results in part from retarded flow of the venous blood out of the brain and in part from secondary œdema of the cerebral substance, in each of them. Both of these lesions, namely, venous hyperæmia and secondary œdema, as we have elsewhere shown, prevent that supply of oxygenated arterial blood from getting into the cerebral capillaries, or reaching the nerve-fibres and ganglion-cells of the brain, which is indispensable for preserving the excitability of the brain itself, and of the entire nervous system. The functional activity of the brain cannot be maintained without the circulation in it of freshly-oxygenated or arterial blood; and whether no blood at all circulates through the cerebral capillaries or merely effete or venous blood, the result is the same, namely, suspension of the cerebral functions. In both apoplexy and epilepsy, then, there is in reality an anæmic state of the cerebral substance; and it is not surprising that in some instances the framing of a diagnosis between them is attended with considerable difficulty. Trousseau, indeed, held that apoplectiform cerebral congestion is, in many instances, really epilepsy; that apoplectic seizures are connected with epilepsy or eclampsia much oftener than is generally supposed, and "that sudden and transient symptoms of apoplexy are in most cases associated with epilepsy or eclampsia." (Vide *Lectures on Clinical Medicine*, vol. i. pp. 19, 22, 38, Sydenham Soc. translation.) In doubtful cases assistance may be derived from inquiring whether the patient has ever had a fit before, whether he has ever experienced the epileptic aura or epileptic vertigo, whether his face, forehead, and neck have ever been covered with small ecchymoses looking like flea-bites, whether his urine was passed involuntarily, and his tongue was bitten during the fit; for when these symptoms are present, they render the diagnosis of epilepsy not only highly probable, but pretty certain. Moreover, the apoplectic condition so often observed in the course of the paralysis of the insane is dependent on eclampsia, just as the analogous condition which follows an epileptic fit is dependent on epilepsy, (*Trousseau ;*) and when softening of the brain suddenly produces the symptoms of coma, it does so by giving rise to epileptiform convulsions or eclampsia. But, when cerebral softening destroys the consciousness without producing epileptiform convulsions, the patient sinks into coma, not suddenly, but gradually, and does not present the phenomena of apoplexy. (See Case XVI., and the comments thereon.) Coma from softening of the brain is therefore not likely to be ever mistaken for apoplexy.

Coma the result of blood-poisoning with urea can generally be distinguished from apoplexy without much difficulty. In one important class of these cases the symptoms of coma do not come on suddenly as in apoplexy, but are gradually developed. The patient sinks by degrees into sopor, lethargy, coma, and carus. (Vide Case X.) In another very large class of these cases the patient is, indeed, suddenly bereft of consciousness, but he also has epileptiform convulsions or eclampsia, which of course show at the first glance that the disease is not apoplexy. (Vide Cases XIV. and XV.) According to my own experience, epileptiform convulsions are generally present in the cases where uræmic intoxication suddenly gives rise to coma. In such cases the attack usually begins with convulsions, and at first there may even be short intervals of consciousness between the paroxysms, as there was in the case of an aged gentleman who came under my observation several months ago; but soon the insensibility becomes continuous, and if the termination be fatal, the convulsive paroxysms usually do not cease to recur to the last. The previous history of such patients generally shows the renal origin of the symptoms; and at the time of the fit either partial or total suppression of the urinary secretion may be present.[1]

But apoplexy of an undoubted character sometimes occurs in patients who have renal disease. It is to such cases that Dr. Thomas Addison, of Guy's Hospital, refers in his paper *On the Disorders of the Brain connected with Diseased Kidneys*, where he says: "This . . form of cerebral affection is that of a sudden attack of coma with stertor, or, in other words, apoplexy; it is, nevertheless, different from ordinary apoplexy; it is the serous apoplexy of authors, and presents the usual general characters of cerebral affection depending upon renal disease; for the face, instead of being flushed, is, in almost every instance, remarkably pale; the pulse, though sometimes small, and more rarely full, is remarkably quiet, or almost natural; the pupil, also, although occasionally dilated or contracted, is often remarkably natural in size, and obedient to light; and there is no paralysis. When the labor of respiration is very great, the general character is apt to be modified by an accelerated pulse, and occasionally by a slight flush of the countenance. The coma is for the most part complete, so that the patient cannot be roused to intelligence for a single moment. The stertor is very peculiar, and in a great measure characteristic of this form of cerebral affection connected with renal disease; it has not, by any means, in general, the deep, rough, guttural, or nasal sound of ordinary apoplexy; it is sometimes slightly of this kind, but much more commonly the stertor presents more of a hissing character, as if produced by the air, both in inspiration and in expiration, striking against the hard palate or even against the lips of the patient, rather than against the velum and throat, as in ordinary apoplectic stertor; the act of respiration, too, is usually, from the first, much more hurried than is observed in the coma of ordinary apoplexy. The peculiar stertor coupled with the pale face has, in more instances than one, enabled me to pronounce with confidence the disease to be renal, without asking a single question, and in cases, too, in which no renal disease whatever had for a moment been suspected." (Vide *A Collection of the Published Writings of the late Thomas Addison, M.D.*, etc., etc., p. 189, Sydenham Soc. ed. London, 1868.) In the cases belonging to this category, however, the apoplectic coma is due, not to poisoning of the blood with urea, but to the effusion of serum into the perivascular spaces of the brain-substance and into the membranes, or, in other words, to cerebral œdema, as we have already shown. This form of cerebral œdema, at least so far as its causation is concerned, appears to be analogous to the kind of pulmonary œdema which not unfrequently occurs with great suddenness in the course of Bright's disease.

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[1] According to my own experience, when coma occurs in cases of uræmic intoxication, it is generally accompanied by epileptiform convulsions or eclampsia. Sometimes, however, it comes on slowly or gradually, commencing in drowsiness, which deepens into stupor and then into coma, without any convulsions. Whenever I have found coma or insensibility *suddenly* produced by uræmic intoxication, it has invariably been attended with epileptiform convulsions; and, in cases where I have seen coma *gradually* induced by uræmic poisoning, anasarca also has generally been present.

When coma unexpectedly appears in a patient who has Bright's disease, we infer that it depends upon cerebral œdema and not on uræmic intoxication: 1. When the fit takes the form of deep coma, with intercurrent eclampsic spasms. 2. When, at the time of its occurrence, the secretion of urine is normal or superabundant in quantity. 3. When the attack is accompanied by marked œdema of the face. 4. When the carotids pulsate strongly during the fit. The last-mentioned symptom is a valuable but often an illy-appreciated one. It generally denotes, as we have elsewhere shown, that the passage of the blood through the cerebral vessels is hindered by some obstruction, such, for example, as compression of the cerebral capillaries, when produced by the effusion of serum into the perivascular spaces of the brain, or by any other cause, would afford. (*Niemeyer.*)

Apoplectiform syncope resulting from fatty degeneration of the heart can generally be distinguished from cerebral apoplexy. It is well known that persons whose hearts have undergone the adipose degeneration or substitution of fat for muscular tissue, to such an extent as to seriously weaken the cardiac contractions, are liable to drop down suddenly in a state of insensibility or syncope, and die on the spot, especially when they are subjected to the shock of injury, or become excited by anger, or depressed by grief and fear. Such cases, on superficial examination, might pass for instances of nervous apoplexy. They can, however, be distinguished from that disorder by their previous history, that is, by having previously shown the symptoms of cardiac enfeeblement or debility, and by exhibiting the phenomena of death commencing at the heart instead of the brain. Such cases are usually attended with well-marked symptoms of syncope. The countenance is pinched, the face and lips are pallid, the respiration is feeble and sighing, instead of being strong and stertorous, the skin is cold, the pulse is frequent, weak, small, irregular, and oftentimes scarcely perceptible at the wrist; and on listening to the heart we find its action characterized by great feebleness and irregularity.

Finally, *apoplexy is to be distinguished from sun-stroke, heat-stroke, or insolatio*, mainly by the history of the case and by the circumstances attending the attack, among which the chief is exposure to the rays of a summer or a tropical sun, or subjection to great heat in some other form. In one important variety of sun-stroke, however, the symptoms are, for the most part, those of syncope, and, therefore, are less likely to be mistaken for those of apoplexy. In another variety of sun-stroke the skin is pungently hot and the body-temperature rises to 104° Fahr., or even to 110° Fahr. (Vide *Wunderlich on the Temperature in Diseases*, p. 132, New Sydenham Soc. translation, London, 1871.) This circumstance added to the history of the attack is sufficient to characterize this affection. There are, however, occasional instances of heat-stroke which cannot be distinguished from apoplexy by any thing besides the history of the attack and the influence which an exalted temperature has obviously exerted in causing it.

Can we make a differential diagnosis of a reliable character between the several varieties of apoplexy? Is there any thing at the time of the fit, or after its more urgent symptoms have passed away, by which we can determine whether the disorder of the brain results from an extravasation of blood, or from an effusion of serum, or from an hyperæmia of the cerebral vessels? And, again, do the morbid manifestations furnish any clue to the seat of the hemorrhage? With reference to the former question, all clinical experience forces us to admit that, in any of the states mentioned, the actual signs may be the same; and that we can never be quite certain of the non-existence of clot. It is true that, when the apoplectic symptoms abate rapidly; when consciousness and thought, however confused, soon return; when the limbs are not paralyzed, or but slightly so, and only for a short time, we have good reason to believe that hyperæmia alone lies at the root of the disturbance; that, in other words, the case is one of those called simple apoplexy. But it is never possible to give a positive opinion, since a clot near the periphery of the cerebral hemispheres may occasion the same phenomena as those specified. And, with regard to a rapid effusion of serum, the difficulty of distinction is quite as great, or even greater. (Vide *Da Costa's Medical Diagnosis*, p. 844.)

With regard to apoplectiform cerebral embolism, however, we can, in many instances, be somewhat more positive in our opinion. Apoplectic coma, with hemiplegia, when suddenly produced in a patient who has valvular disease of the heart, is almost certainly due to embolism of the cerebral arteries, and not to extravasation of blood in the brain; for apoplectic coma, in the classical sense of the term, is but seldom produced by cerebral hemorrhage, and hemiplegia, as well as coma, when produced by cerebral hemorrhage, is, for the most part, developed not all at once or suddenly, but more or less gradually, as we have elsewhere fully shown. The presence of valvular disease of the heart strengthens the diagnosis very much in a case that is suspected to be cerebral embolism, because it shows us at once the place from which the embolia may have been washed away by the current of the circulating blood, and carried into the cerebral arteries.

There is still one symptom remaining which is worthy of special mention in connection with the diagnosis of apoplexy, and that symptom is throbbing of the carotid arteries. If these arteries pulsate strongly when the left ventricle of the heart is free from hypertrophy and the other arteries do not pulsate abnormally, it is a sign, not that there is "a rush of blood to the head," but that the flow of blood through the brain is obstructed by some mechanical impediment. In cases of cerebral embolism the passage of blood through the brain is obstructed by the plugging up of some of the cerebral arteries with embolia; in cases of cerebral hemorrhage the same result is produced by a compression of the capillaries in the cerebral substance, which is effected by the pressure that arises from the extravasated blood; in cases of serous apoplexy the flow of blood through the brain is impeded by a compression of the cerebral capillaries which results from the effusion of serum into the perivascular spaces and into the membranes of the brain; and in cases of congestive apoplexy, by retardation in the escape and consequent stagnation of the venous blood in the cerebral vessels, together with collateral œdema of the cerebral substance. This symptom is also induced by any pathological state of the brain or its membranes which encroaches upon or lessens much the intracranial space, and therefore it is not characteristic of apoplexy; but, in doubtful cases, its presence or absence may prove of considerable importance in settling the diagnosis of that disease.

With regard to employing the *ophthalmoscope* in the diagnosis of apoplexy and cerebral hemorrhage, Dr. Allbutt, in a recent work, says: "In cases of recent hemorrhage, taken simply, we have but little need for the ophthalmoscope. In a few cases of heavy effusion, I have seen a certain amount of venous distention in the retina, either with or without some slight serous effusion also. M. Bouchut gives a number of cases of hemorrhage, and speaks of such changes in the disk as being very frequent. The cases are by no means satisfactory ones. In many there was no autopsy, and in investigations of the present kind, cases without autopsies are really without value. . . . In other cases where an autopsy was made, no account is given of the state of the kidneys. In one or two instances, M. Bouchut's descriptions of the signs in the eyes are very suggestive to me of the first stage of retinitis albuminurica, a condition very likely to appear in such subjects. . . . It would have been at least desirable to state whether albumen was present in the urine or not." (Vide *Allbutt On the Use of the Ophthalmoscope in Diseases of the Nervous System, and of the Kidneys*, London, 1871, p. 178.) Again, Dr. Hughlings Jackson remarks on the same point: "I have once, in a case of extensive meningeal hemorrhage (which I saw at the Hospital for the Epileptic and Paralyzed, ...) seen extreme dilatation of the retinal veins, *but in most cases of cerebral hemorrhage I have found nothing to call abnormal*." (*Ibid.* p. 179.) Finally, Dr. Allbutt observes: "Hemorrhage into the retina has been recorded in several cases of encephalic hemorrhage, but I am not disposed to think that any pressure upon the recurrent vessels could burst the veins of the retina unless these were themselves conspirators. In fact, retinal hemorrhages, independent of albuminuric retinitis, are not uncommon in old people in whom there is no cerebral hemorrhage and in whom there may be none." (*Ibid.* p. 180.)

The last-named writer also says: "The frequent occurrence of albuminuric retinitis and encephalic hemorrhage in the same person is, however, reason enough to urge us to examine the retina in all cases of encephalic hemorrhage. The discovery of retinitis or its traces should make us give a far graver opinion than in a case where there was no degeneration of the kidney, and a somewhat graver prognosis than in cases where albumen in the urine existed without retinitis. It may be the chances of individual experience which lead me to say this; but it has certainly happened to me very often to see retinitis in apoplectics who have been rapidly cut off by a second attack, while others having albuminuria, but not retinitis, have survived much longer. I may extend this remark further, and say that I believe a person who has not suffered from apoplexy, but who has retinitis, is in great danger, though in adding this I am going beyond my present subject. Perhaps I am not exceeding my limits, however, in calling attention to a disorder of vision, not attended with any visible change in the eye, which is often to be noticed in patients suffering from a mesocephalic hemorrhage. This disorder is hemiopia, and depends, no doubt, on the distending pressure of the clot upon one optic tract, for it passes off with the absorption of the effusion. Such patients have satisfactory central vision, but they may be seen to hesitate and grope after objects lying on one side, the dark side, of course, being the side opposite to the hemiplegia. This functional disorder—for it seems [almost] never to advance to atrophy—may remain for some time and be troublesome, but I think it [almost] always disappears as the pressure is ultimately removed." (*Ibid.* pp. 181, 182.)

"There is another change I have often seen in the eyes of those struck down with [cerebral] hemorrhage, and that is a certain degree of nerve-atrophy. This atrophy is more than mere senile pallor of the disks; it is an actual though incomplete atrophy, and is attended with some degree of loss of vision, though this may not be sufficient to demand serious attention. The disks look white and diminished, and the vessels are fine. I have not been able to ascertain the state of the field of vision in these cases, but this would be an important addition to our knowledge if obtained. I believe that the atrophy is due to atheroma of the encephalic arteries, and thus it may in some degree be recognized as a sign of atheroma, and as a forerunner of apoplexy." (*Ibid.* p. 182.)

With regard to the miliary aneurisms which so often form on the minute branches of certain cerebral arteries and lay the foundation for cerebral hemorrhage, M. Henry Lionville (*Archives Générales de Médecine*, April, 1870, p. 503) appears to have noted the coëxistence of miliary aneurisms in the eye with the above-mentioned, as early as 1868. He now describes the case of a patient, aged 72, who died in the Salpêtrière after several small extravasations. Innumerable miliary aneurisms were found in the cerebrum, the cerebellum, the pons, and the membranes. There were also miliary aneurisms in both retinas, some of which corresponded to small hemorrhages in the retinal layers. Examined under the microscope, these aneurisms presented a marked likeness to the miliary aneurisms of the encephalic arteries. The report does not state whether the ocular aneurisms were discovered by the mirror; apparently they were not found until the post-mortem examination. Dr. Allbutt, however, says that his search for miliary aneurisms in the eye in similar cases has been fruitless. (*Ibid.* p. 183.)

"If we leave recent apoplexies, [hemorrhages,] and turn to ancient ones, we are no longer in want of curious ophthalmic signs. It is a very remarkable and interesting fact that old clots do often give rise to both neuro-retinitis and atrophy, though it is very uncertain how this comes about. Of course it is easy to set neuritis down to 'irritation,' propagated by a 'foreign body' lying long in the brain; but we have very little evidence in favor of our accepting this explanation in its only intelligible sense, in the sense that it is a creeping proliferative process." (*Ibid.* p. 183.)

Concerning the employment of the *ophthalmoscope* in diagnosing cerebral embolism, Dr. Allbutt also says: "*Embolism* does not give rise to any definite eye-mischief, so far as I know, unless it be followed by extensive central softening. The contrary is stated by some writers, and I myself thought it likely that immediately after the accident, when the internal carotid was propelling blood over an area suddenly diminished, there might be some evidence of increased arterial tension, either in dilated vessels or in some degree of effusion. In one or two cases I have noticed some degree of haziness about the borders of the disk and vessels, *but nothing that I could with a good conscience call a distinctly morbid state.*" (*Ibid.* p. 184.)

Dr. Allbutt, however, states, from his own experience and that of others, that embolism of the arteria centralis retinæ, when the occlusion is sudden and complete, is attended with the following ocular phenomena: "1. Instantaneous, or almost instantaneous, loss of function. 2. Sudden emptiness of the arteries and capillaries, and more or less of the veins. 3. Œdema of the parts deprived of supply, which œdema comes on gradually, and, in cases seen early, amounts to little more than a slight haziness around the yellow spot. 4. A tendency in the later stages to hemorrhage from collateral vessels, (peripapillary and choroidal.) 5. Ultimate thickening of the adventitia of the vessels and fatty degeneration of the retinal tissue, with deposit of cholestearine." . . . . . . "I think," he says, "we may fairly assume that the changes in the encephalon are of the same kind, namely, sudden emptiness of the arteries and corresponding venules, with some momentary deficiency in the contents of the sinuses, œdema of the affected parts, tendency to collateral hemorrhages, and instantaneous loss of function." (*Ibid.* p. 293.)

Dr. Allbutt further says: "*Softening* of parts of the encephalon other than the optic centres, tracts, or nerves, without sclerosis, is not attended with any significant changes in the eye." (*Ibid.* p. 184.)

3. Prognosis of Apoplexy.—Hippocrates, as we have elsewhere stated, declares: "It is impossible to remove a strong attack of apoplexy, and not easy to remove a weak attack." On this aphorism Dr. Adams, the translator, remarks that the experience of twenty-two centuries affords

no reason to call in question the prognosis here announced. It is thus rendered by Celsus: "Isque morbus mediocris vix sanatur, vehemens sanari non potest." (Vide *Works of Hippocrates*, p. 713, *Aphorisms*, sec. ii. 42, Sydenham Soc. ed., London, 1849.)

Apoplexy is, generally, more dangerous in proportion as the respiration is stertorous, the deglutition difficult, and the pulse disordered. Each succeeding attack is also more dangerous than the former. Popular opinion (and it is useful in pronouncing an opinion to know what are popular beliefs) supposes that the patient may suffer three apoplectic attacks, of which the first is light, the second is followed by paralysis, and the third proves fatal. This number is exceeded in only a few instances. (*Aitken.*)

From the congestive form of apoplexy the recovery may be rapid and complete, provided that a judicious plan of treatment is employed. Cases of this affection which are apparently hopeless sometimes recover in a remarkable manner. I remember one striking instance of that sort. Many years ago, I was called to a Mr. L——, aged about seventy years, who lay in a profound fit of congestive apoplexy. He looked much older than seventy, and had been in the habit of drinking to excess for many years. He was utterly insensible, his pupils were contracted, conjunctivæ injected, face deep-red and turgid with blood, carotids pulsating strongly, respiration deeply stertorous, and radial pulse slow, full, and strong. I was informed that the attack came on suddenly, and that he had already lain insensible, and just as I then saw him, for more than twenty-four hours. I gave a very unfavorable prognosis, so much so that his wife told me I need not call again. To my great surprise, he recovered completely, and lived more than fifteen years afterward, dying at the age of eighty-six. By the way, the treatment consisted mainly in the application of ice to his head, and in the administration of brisk purgatives. But congestive apoplexy very often proves fatal, as the cases related in Chapter IV. abundantly show, and we should always be careful in regard to holding out hopes of recovery from it.

Sometimes, when the sight has been destroyed by apoplexy it is recovered again, in a remarkable manner, after much time has elapsed. Forbes Winslow mentions the following case: A gentleman, by reason of an apoplectic seizure from which he recovered, lost his sight and continued blind for about seven years. After this period, while one day out in his carriage, he suddenly recovered his vision. It was afterward found that he still entirely retained his skill in drawing, for which he had been much distinguished before he became blind. (Vide *Obscure Diseases of the Brain*, etc., p. 427.)

For the prognosis of sanguineous apoplexy, see *Prognosis of Cerebral*

Hemorrhage in Chapter V.; and for the *prognosis* in cases *of Apoplectiform Cerebral Embolism*, see Chapter VI.

4. Treatment of Apoplexy.—For the purpose of securing convenience and clearness in description we shall discuss this topic under each of the following heads: 1. What is required before the attack; 2. What is required during the attack; 3. What is required after the attack.

1. *On the treatment to be employed before the attack of apoplexy.*—It is prior to the attack that the physician can do most for the benefit of apoplectics or those who are liable to this disease. If the premonitory symptoms of it be allowed to go unheeded, if the precautionary measures which they may indicate be not promptly adopted, many will perish of this terrible disorder who might otherwise be saved. In such cases the grand object of the treatment to be employed should be to prevent the attack. So long as that is done the patient remains comparatively safe. At this period the physician's duty mainly consists in seeking out and removing, or rendering inert, the various causes which in each case tend to bring on the fit of apoplexy. In no other disease are the causal indications, as they are sometimes called, of more importance than in this; and in but few are they of equal importance. Among adults, the chief sources of apoplexy are the abuse of food, the abuse of drinks, the abuse of narcotics, and excessive brain-work, as we have already shown in Chapter III. These causes are all preventable, and, for the most part, are under the control of the patients themselves. At all events, the physician cannot hope to render these causes of apoplexy nugatory, unless he has the hearty coöperation of the patient and his attendants. Hippocrates, in his first Aphorism, wisely declares *inter alia*: "The physician must not only be prepared to do what is right himself, but also to make the patient, the attendants, and externals coöperate." (Vide *Works of Hippocrates*, p. 697, Sydenham Soc. ed., London, 1849; see also the motto of this volume.) The physician whose duty it is to treat such cases of threatened apoplexy as are referred to above should, first of all, endeavor to secure the cordial coöperation of the patient in thoroughly changing his mode of life. When apoplectic warnings have once occurred, the hard-drinker must give up his liquors, his wines, his ale or beer, and take to using water instead thereof, the gourmand must abandon his luxurious meals and restrict himself to plain fare, the opium- or hashish-eater must abstain from his enchanting drugs, and the brain-worker must give his overtasked mind the repose it so much needs. Likewise if the patient has any other habit which tends to produce the disease in question, he should be counselled to abandon it without delay.

But there are certain morbid states of the organism itself which also serve to induce paroxysms of apoplexy. These, too, demand the physician's attention, in order that he may procure their removal, or limit their pernicious influence. Among these morbid states which are provocative of apoplexy, we have first to speak of general plethora, or a state of general turgescence of the vascular system, whose symptoms have already been described. Its treatment must consist in the adoption of a restricted diet, or in the employment of non-nutritive substances; in the avoidance of beer and all other alcoholic drinks; in lessening the hours devoted to sleep; and in the use of active exercise in open air. Saline purgatives also often do good. The bromide of ammonium in doses of ten grains three times a day may, not unfrequently, prove useful. In extreme cases the abstraction of blood ought to be had recourse to; the quantity to be taken being measured by the immediate effects. Venesection judiciously employed will often afford sensible relief; and provided the intervals between its use are not too short, it is difficult to see what harm can result therefrom. (*Tanner.*)

We must next consider hyperæmia of the brain, and its treatment, in the same connection. We have elsewhere shown that cerebral hyperæmia is the stepping-stone to apoplexy in a large majority of the cases where that disease occurs. Its treatment, therefore, is of corresponding importance to nearly all of those who are threatened with apoplectic strokes. Indeed, the treatment of apoplexy is to a great extent the treatment of cerebral hyperæmia. Prominent among the measures recommended for the relief of hyperæmia of the brain are blood-letting, both general and local, the application of cold to the head, and the administration of purgatives and other derivatives. But we should not use one or other of them indifferently, nor employ all of them at the same time, in every case that occurs. We should first ascertain the causes which have produced the cerebral hyperæmia, and then be guided by the indications which they afford, in selecting the remedial measures to be employed for its relief.

When hyperæmia of the brain has resulted from long-continued abuse of alcohol or of narcotics, or from excessive mental excitement, nothing useful is to be expected from general blood-letting, and large venesections often prove injurious in such cases; on the other hand, the indications are to apply the ice-bag or frozen compresses to the head, to administer derivatives, and to give the bromide of potassium or sodium also, if there is wakefulness, restlessness, or much excitability. Among the derivatives commonly employed are mustard pediluvia, sinapisms to the epigastrium and extremities, and strong purgatives. In acute and threatening cases of these forms of cerebral hyperæmia croton-oil has a high repute, but, in chronic cases, pills of aloes, colocynth, gamboge, jalap, etc., are usually prescribed. In acute cases, blisters also applied to the nape of the neck, and in chronic ones, the establishment of an issue in the arm, often do much good. Sometimes, and especially in cases where old hemorrhoids have suddenly ceased to bleed, the application of leeches about the anus has a wonderfully beneficial effect. In a case of chronic cerebral hyperæmia due in part to mental excitement, and in part to the abuse of narcotics, I have lately seen much good done by the administration of arsenious acid in doses, or granules, containing each gr. $\frac{1}{26}$, taken three times a day, after meals. With regard to the employment of arsenic for the relief of cerebral hyperæmia, we may add that Lamare-Picquot strongly recommends the prolonged use of this agent as an effectual means of subduing congestion likely to give rise to apoplexy. (Vide *New Sydenham Soc. Year-Book*, 1860, p. 414.) Again, the same writer administers arsenious acid in doses up to about one fifth of a grain per day, in all cases where he has reason to believe that the red globules are in excess in the blood. When they amount to more than 54 in 100 parts, signs of cerebral congestion ensue. By the use of arsenic the globules are diminished. (Vide *New Sydenham Soc. Year-Book*, 1861, p. 162.) Finally, M. Lisle, Director of the Marseilles Lunatic Asylum, thus terminates a paper, which he read to the Academy of Medicine, upon the "Treatment of Cerebral Congestion and Hallucinations by Arsenious Acid:"

1. The insane frequently present more or less distinct signs of cerebral congestion, and the subjects of hallucination always do so. In 193 cases of the latter description treated by arsenious acid, 131, or 67 per cent, were cured, and 29 experienced marked and durable amelioration.

2. Hallucination, considered heretofore as a symptom of insanity, is really only a complication, almost always of serious import. It is the most characteristic symptom of cerebral congestion, the essential nature of which is little known, and which may terminate in insanity, although this is not a necessary consequence.

3. Arsenious acid is truly a specific remedy in this affection. It is also of great utility in paralysis, incoherency, and melancholy unattended with hallucinations, but presenting symptoms of cerebral congestion.

4. Administered with prudence and carefully watched, it is one of the most inoffensive agents of the materia medica. The dose should vary from 5 to 15 milligrammes, administered three times a day, just before each meal. (Vide *Half-Yearly Abstract of the Medical Sciences*, vol. xlvi. p. 41; also *Medical Times and Gazette*, Sept. 28, 1867.)

In cases where *hypertrophy with increased action of the heart* and coincident diminution of resistance in the blood-vessels of the brain have induced hyperæmia of that organ, all mental excitement, all immoderate bodily exertion, and the use of all stimulating drinks, must be sedulously avoided, for they increase the heart's action, and still further distend the already overcharged arteries. Such patients must also beware of immoderate eating and drinking, in order to avoid the plethora which, although but transient, always follows upon a free use of food or drink. How frequently the long-threatened fit of apoplexy occurs in the midst of the plethora which has been developed after a long and hearty meal! In such cases we should bleed as soon as apoplectiform cerebral congestion seems imminent; for in them venesection cannot safely be replaced by applying the ice-bag to the head or leeches behind the ears.

In those cases where collateral determination of blood to the brain has occurred, we must first of all attempt to remove the obstructions to the circulation by which the pressure of blood in the carotids is increased. Evacuating the intestines by laxatives, or by enemata of vinegar and water, often has a marvellous effect, which is induced by nothing else, both in adults who are constipated and suffering from headache, tinnitus, dizziness, etc., and especially in children where constipation is accompanied by convulsions, etc. If these measures prove insufficient and symptoms of cerebral oppression occur, from which we apprehend danger, it is proper to draw blood here also, in adults by venesection, in children by leeches to the head. (*Niemeyer.*)

When passive hyperæmia of the brain results from compression of the jugular veins or vena cava descendens, or from heart and lung-diseases, venesection or leeching behind the ears may be employed, if the obstruction to the flow of blood cannot be removed. We have already shown that venous congestion of the brain by arresting the supply of arterial blood to the cerebral substance diminishes or suspends the excitability of the nerve-fibres and ganglion-cells belonging to the organ, and produces cerebral depression and cerebral paralysis. Now, the greater the freedom of escape from the brain which we give to the venous blood in such cases, the sooner we shall succeed in removing the symptoms of depression and paralysis. This may be done by the application of leeches behind the ears, for by it the tension is lessened in the veins outside of the skull, with which the emissaria Santorini communicate; or by venesection, by which the tension in the anonymous veins is diminished, because less blood enters them from the arm. In such cases we cannot expect any benefit from the application of cold to the head, nor from the employment of purgatives and blisters. (*Niemeyer.*)

In cases where hyperæmia of the brain results from too much nourishment or a state of general plethora, instant diminution of the quantity of blood may be urgently indicated, and a well-timed venesection not unfrequently prevents a threatening apoplexy. It is very important to regulate the mode of life of such patients, to show them the danger of prolonged and luxurious meals, to let them eat little, drink water instead of wine, and walk a great deal. Of course, in each case, peculiar circumstances will require some deviation from, or modification of, the plans of treatment advised, and different directions as to regimen. (*Niemeyer.*)

We should be more cautious about employing venesection in cases where disease of the heart coëxists than in other cases, and should not have recourse to it in such cases any oftener than absolutely necessary, for it is almost certain that the practice of bleeding favors degeneration of the heart; and attenuation of the blood undoubtedly promotes the tendency to dropsy. (*Niemeyer.*)

We must also briefly state the plan of treatment which Bright's disease requires when it threatens to produce an attack of apoplexy. In such cases the indications are to lessen the renal congestion, to promote the excretion of urea, and to increase the flow of urine. The first indication requires the application of dry cups to the loins, and the administration of belladonna or atropia;[1] the second, either purgation with elaterium, or the production of profuse diaphoresis with the infusion of ipecacuan.; and the third the administration of digitalis. It is often advisable to combine the belladonna and digitalis thus:

℞. extract. belladon. (opt.) grs. vi.
pulv. digitalis (opt.) grs. xxiv.
mucilag. gum. tragacanth. q. s. ut
ft. pil. No. 24.
S. Take a pill night and morning.

We must also bear in mind the influence which atmospheric temperature and the seasons exert in the production of apoplexy, while conducting the treatment of those who are threatened with this disease. Extremes of temperature act as powerful excitants of apoplexy; for cold drives the blood from the surface of the body upon the internal organs, and heat impairs the tone of the capillaries and tends to produce vascular turgescence and passive congestion. Sudden and great changes in the temperature of the atmosphere also tend strongly to produce apoplexy in those who are already predisposed to its occurrence, for they rapidly exhaust the nervous power. We should therefore advise all patients who are threatened with apoplexy not to expose themselves to extremes of temperature whether in summer or winter, and to protect themselves as much as possible against sudden changes in the weather.

Finally, veratrum viride may sometimes be administered with benefit in cerebral hyperæmia, and we must now state the circumstances under which it may be so employed. In a case of so-called "rush of blood to the head," or active determination of blood to the brain, caused in part by over-stimulation with alcoholic drinks, in part by undue mental excitement, and in part by abnormal activity of the heart, which strongly threatened to produce an apoplectic fit, and in which venesection appeared to be inadmissible, I administered Norwood's tincture of veratrum viride and applied ice to the head with very satisfactory results. The heart's action and the vascular excitement generally were quickly reduced to the normal standard, and the threatening symptoms all soon passed away. I think that in similar cases veratrum viride, combined perhaps with bromide of potassium, may generally be administered with much advantage.

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[1] Extract belladon. in doses of gr. ¼, or atropia in doses of gr. $\frac{1}{60}$, taken once or at most twice a day, has, in my hands, proved a most useful remedy for renal congestion.

2. *On the treatment to be employed during the attack of apoplexy.*— The physician, if consulted in season, can generally succeed in warding off or arresting a threatened attack of apoplexy by the prompt employment of an energetic and still judicious plan of treatment. But after the stroke has occurred he is comparatively powerless to benefit his patient. As Hippocrates justly observes: "It is impossible to remove a strong attack of apoplexy, and not easy to remove a weak attack." The only time, then, for really successful interference on the part of the physician with this dreadful disease, is while the attack is in process of formation. After the attack has begun he is, in most cases, practically limited to combating the more dangerous symptoms, or, in other words, to obviating the tendencies to death.

For the relief of apoplectics blood-letting and purgatives have, in all ages, been recommended. Celsus says: "His sanguis mittendus est; veratro quoque, vel alvi ductione utendum." (Vide *Celsus de Medicina*, Liber III. Cap. XXVI.) But the employment of venesection should be much more restricted after the attack has occurred than prior thereto. Observation has shown that it is seldom admissible in cases of apoplectiform cerebral hemorrhage, (vide *Treatment of Cerebral Hemorrhage*, in Chapter V.,) that it is almost never called for in cases of apoplectiform cerebral embolism, that it should never be employed in cases of serous apoplexy, and but seldom, comparatively speaking, in cases of congestive apoplexy. We should generally be very cautious in regard to the employment of venesection even in cases of congestive apoplexy. Usually it does no good; sometimes it obviously does harm in such cases. In Case XIX., and likewise in several other instances which we have related, it did no good whatever. In Case XX, it seemed to do harm, for it made the pulse more frequent and feeble; while Cases XXI. and XXII. recovered without the abstraction of any blood. Besides, even when the apoplectic fit is cut short by venesection, it happens not unfrequently that the patient never recovers from the consequences of the general bleeding itself, because he is too much weakened in constitution by excesses in living to stand it. A case of that sort is mentioned in the latter part of Chapter IV. The coma was very profound and the symptoms very alarming. They were promptly relieved, however, by bleeding from the temporal artery, but the man died a few days afterward of chronic alcoholism, as it was said. He never recovered from the depression which followed the abstraction of blood. Although it relieved the apoplectic symptoms, it did not in reality increase the chances of his getting well. But venesection may sometimes be employed with advantage in cases of congestive apoplexy. If the patient is comparatively young and vigorous, if he is not much broken down by alcoholic intemperance and other excesses in living, if the pulse is full and strong, the heart-sounds clear and regular, the coma deep, the stertor loud, and the determination of blood to the head obvious, with no signs of pulmonary œdema present, then the cautious abstraction of blood may be productive of much benefit. At the same time, however, it should be remembered that venesection cannot remove the consecutive œdema of the cerebral substance and cerebral membranes which occurs so often with disastrous consequences in cases of congestive apoplexy, (vide *Cases* III., IV., V., VI., VII., VIII., IX., etc., and *the résumé of the Cases near the end of Chapter* IV.;) also that venesection generally fails to set the blood in motion again when it has become stagnant in the cerebral vessels, as it does in cases of apoplexy from passive hyperæmia of the brain; and that for these reasons, among others, blood-letting to be effectual in such cases must be employed before the fit begins. Thus, it appears that great skill and delicacy of judgment are sometimes required in order to decide correctly whether general bleeding should be used for individual cases of apoplexy. In doubtful cases, however, local bleeding by cups or leeches, applied to the temples and nape of the neck, may not unfrequently be practised with advantage.

Purgatives also have been employed in the treatment of apoplexy from the earliest times, as is shown by Celsus' recommendation quoted above, and they continue to be in good repute. This class of remedies is applicable to nearly all the cases that come under treatment, for it is but seldom that the debility is so great as to contraindicate the use of at least a laxative. Much good is often done to apoplectics by this class of remedies. Among the most useful of them for such patients are compound extract of colocynth, croton-oil, and castor-oil. In cases of *apoplexie foudroyante*, or thundering apoplexy, as it is sometimes called, it is often advisable to administer strong enemata for the purpose of securing more speedy action of the bowels.

Emetics have been much esteemed as remedies for apoplexy by some distinguished practitioners. For example, they were recommended by

Sydenham.[1] The administration of one will generally prove useful at the commencement of an attack, especially when the stomach is overloaded, as, for example, with a hearty meal that has just been taken. Under such circumstances the prompt evacuation of the stomach usually affords no inconsiderable amount of relief to the overloaded brain. Indeed, overfulness of the stomach is generally reckoned as one of the more important of the exciting causes of apoplectic fits. The following is a convenient and at the same time safe prescription. ℞. pulv. ipecacuan. grs. xx., vin. ipecacuan. f. ℥ j. M. To be given at a dose.

Repellents and revulsives often do good in cases of apoplexy. Among the repellents the most efficacious are cold affusion or the pouring of cold water on the head, and the application of the ice-bag or of frozen compresses to the same part. The value of cold affusion of the head in lethargic cases was known to the ancients. Celsus says: "Excitat autem validissime repente aqua frigida infusa." (Vide *De Medicina*, lib. iii. cap. xx.) Cold water does, indeed, rouse most powerfully when poured on suddenly. Dr. West strongly recommends cold affusion of the head for the relief of cerebral congestion occurring in infants and children. He says: "When you have determined to resort to it, the child must be taken out of bed, wrapped in a blanket, and laid upon the nurse's lap, with its face downwards, while you pour a stream of water from a little height upon its head. The most effectual way of doing this, though one not always practicable, is to place the child under the cock of a water-cistern, or the spout of a pump, since you can then continue the stream uninterruptedly for five or six minutes. I have seen remarkable instances of convulsions arrested, and of children aroused from coma, by this means; but you must bear in mind that the agent is one of great power, and you must feel the pulse from time to time during its employment, lest you should, by its long continuance, produce too great a depression of the vital energies." (Vide *Lectures on the Diseases of Infancy and Childhood*, p. 52, Am. ed. 1868.) In our own experience, cold water was poured on the head of an adult having apoplexy, with great benefit, in Case XXI., and ice was applied to the head with decided advantage in Case XXII., and in the old gentleman's case mentioned a little way back. When it is deemed advisable to use frozen compresses, they may readily be prepared by placing wet cloths, properly folded, beneath a vessel containing a mixture of salt and pounded ice or snow. But, even in adults, when we pour cold water on, or apply the ice-bag or frozen compresses to, the head, we should watch the effect produced on the pulse, in order to discontinue or moderate them if the resulting depression becomes too great. The effect of these repellents can be more or less considerably increased in patients who are not bald, by cutting off the hair or shaving the head. When cold affusion, or the application of the ice-bag or of frozen compresses to the head, does good in cases of apoplexy, the livid hue and swollen or congested appearance of the face gradually subside, for the vaso-motor nerves are roused again into activity, the swollen or dilated blood-vessels contract upon their contents, and thus the stagnant blood is again set in motion. The effect which we see produced on the circulation of blood in the face, under such circumstances, is doubtless also produced simultaneously on the circulation of blood in the brain within. If this view be correct, and we see no good reason to doubt it, the repellents above mentioned are the most valuable of all the means we possess for treating the so-called congestive forms of apoplexy. Moreover, in proportion as the appearance of the face becomes natural under the use of these repellents, the stertor grows less loud, and the other symptoms of coma disappear. As happened in Cases XXI. and XXII., which are related in Chapter IV.

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[1] Vide *Sydenham's Works*, vol. ii. p. 259.

Again, the advantages derived from cold affusion, and the application of the ice-bag or frozen compresses to the head, in cases of apoplexy, may be supplemented by the simultaneous use of revulsives or derivatives; such, for example, as the purgatives and enemata above mentioned, dry cups put on the temples and neck, and sinapisms, but especially the latter. For adults, these should be made of full-strength mustard-powder, and should be applied to the epigastrium, nape of neck, inside of thighs, calves of legs, and ankles. Furthermore, sinapisms are applicable to cases of apoplexy where the debility is too great to allow the employment of cold affusion, or the ice-bag, or frozen compresses. In grown people, they may be kept on a long time, when necessary, without danger of producing bad consequences.

Aconite in moderate doses also seemed to do good in Case XXII.; at all events, it did no harm that was perceptible, and theoretically it seems capable of doing good in similar cases.

In treating apoplexy, T. Inman gives an emetic if the stomach be loaded. If the face be congested, he resorts to local bleeding. If the heart be hypertrophied and contracting strongly, he practises a small venesection. If none of these conditions are present, the sole indication is to sustain the vital powers, and restore, if possible, the cerebral circulation. (Vide *New Sydenham Soc. Year-Book*, 1862, p. 91.) For the purpose of sustaining the vital powers, it will be necessary to administer nutrients if the patient lies long in the fit. They must be in a liquid form, in order to be swallowed or to be poured into the stomach through an œsophagus-tube. The best articles are beef-tea, milk, rice-water, and barley-broth.

It not unfrequently is also necessary to give alcoholic stimulants, such, for example, as wine and brandy, for the purpose of obviating the tendency to death.

The *treatment of nervous apoplexy* should be conducted in a manner somewhat different from that of congestive apoplexy. The disease essentially consists in an anæmic state of the brain-substance and its constituent parts, that is suddenly produced by the operation of emotional causes, such as excessive joy, terror, grief, and despair. The symptoms which attend it bear considerable resemblance to those of syncope and surgical "shock," and it should be treated in about the same way. The patient should be placed in a horizontal posture, with the head lying low, diffusible stimulants should be administered as restoratives, and sinapisms should be applied to the epigastrium, arms, and legs. For diffusible stimulants we may employ spt. ammon. aromat., spt. ætheris comp., or spt. vini Gallici, suitably diluted with water, and administered *per orem et per anum*, according to the severity of the case.

When *apoplexy occurs in children* it should be treated on the same general principles as the corresponding variety of the disease when it occurs in adults. It should, however, be stated that, when congestive apoplexy in a sthenic form presents itself in children, the abstraction of blood is much more likely to prove beneficial than when it presents itself in adults. We can probably find an explanation of this difference in the fact that the cerebral blood-vessels and cerebral substance have not yet become altered by disease in children, as we generally find they are in adults who die of congestive apoplexy. (See *the Summary of the Cases* in latter part of Chapter IV.)

Apoplectiform phenomena are sometimes produced in *children* by thrombosis of the cerebral sinuses, as we have already shown in Chapter VIII. In such cases the thrombosis itself generally results from profuse diarrhœa, marasmus, or some other form of exhausting disease. We can do but little, if any thing, for the relief of this kind of cerebral disturbance.

When apoplectiform phenomena, unconnected with thrombosis of the cerebral sinuses, occur in pale-looking, badly-nourished *children*, or in those emaciated from marasmus, as they did in Cases XLVI., XLVII., and XLVIII., a stimulating and supporting plan of treatment is indicated, and the free administration of such remedies as brandy, milk-punch, and beef-essence. By the prompt and liberal use of stimulants, such patients can sometimes be saved, but the prospect of their recovery is generally but slight.

3. *On the treatment to be employed after the attack of apoplexy.*—We cannot say a great deal on this topic, but most of it is of great importance to the patient, or rather it is of great importance for him to heed it. The man who has had one attack of apoplexy, and escaped with his life, is strongly predisposed to have another, which will use him still more severely. Such a one, therefore, should ever afterward be very careful to avoid every thing which tends to overfill or distend the cerebral blood-vessels; that is, every thing which tends to produce cerebral hyperæmia. He should lead a regular life, drink water instead of wine,[1] and take plenty of exercise in the open air. He should keep his bowels regular by the use of suitable food, and laxative medicine also, if necessary. He should be especially careful to avoid long, luxurious meals, excesses in drink, and excesses in venery. The physician must also insist upon issuing the most precise orders for the guidance of such patients, and upon exactly regulating the quantity and quality of the meals of which they are to partake. Niemeyer says: "In this connection I may mention an act of folly which I have often seen practised by tavern-keepers and itinerant wine-dealers. The latter often suppose that, by a free use of water, they can counteract the pernicious influences to which they expose themselves, although it is evident that the plethora arising from a full meal would only be increased by an immoderate addition of liquid." The same writer also says: "These patients often do well under the use of the *whey-cure.* The *grape-cure* also has a good effect, but not unless we restrict the supply of other kinds of food. If we permit the patients to take their ordinary meals, and then to eat daily three or four pounds of grapes besides, danger may easily arise, particularly that of congestion of the brain and apoplexy. I once saw a fresh attack of apoplexy occur in a person who, after having been four weeks at Marienbad, where he had done very well, was then daily eating four pounds of grapes at Vevay, as an *after*-treatment, without, however, making any reduction in the amount of his other food." (Vide *Niemeyer's Text-Book of Practical Medicine*, vol. i. pp. 314, 315, Am. ed. 1870.)

Sometimes the patient exhibits symptoms of cerebral irritation for a while after coming out of an apoplectic fit, and it may be advisable to use counter-irritation and sedatives for its relief. This occurred in Case XXII. A blister was drawn on the back of his neck, and tinct. rad. aconiti was continued in moderate doses until all signs of cerebral irritation ceased. Compresses wet with cold water were substituted for the ice-bag as an application to his head, his bowels were kept open by laxatives, and a milk-diet, with beef-tea and eggs, was allowed. This patient made an excellent recovery. Blistering was also recommended by Sydenham in the treatment of apoplexy. (Vide *Sydenham's Works*, vol. ii. p. 259.)

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[1] This is an ancient maxim in the treatment of apoplexy, for Celsus says, "a vino abstinendum." (Vide *De Medicina*, liber iii. cap. xxvi.)

For an account of the treatment appropriate for *Cerebral Hemorrhage*, see Chapter V.; and for that of *Cerebral Embolism*, see Chapter VI.

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CHAPTER XI.

ON EPIDEMIC CEREBRO-SPINAL MENINGITIS, OR SO-CALLED CEREBRO-SPINAL FEVER, SPOTTED FEVER, ETC.

Synonyms.—Definition.—The first outbreak on record referred to.—Many others mentioned.—*Nature of Epidemic Cerebro-Spinal Meningitis;* it is not an idiopathic fever; the proof given.—*Case* LVII. Epidemic cerebro-spinal meningitis; at first several paroxysms of intense pain, located in the spine between the shoulder-blades, occurred; then loss of consciousness, coma, and death suddenly ensued; there were no symptoms of fever whatever; autopsy.—*Case* LVIII. Epidemic cerebro-spinal meningitis; first, paroxysms of extreme restlessness occurred; next, convulsive movements of an irregular character appeared; then delirium, with screaming and much tossing in bed, but without any signs of fever, were present; finally, stupor, with dilatation of pupils, inability to swallow, coma, and death occurred; autopsy.—*Case* LIX. Epidemic cerebro-spinal meningitis; the first symptoms noticed were debility and loss of appetite; next, headache, nausea, thirst, feverishness, and restlessness; then abnormal heat of head, injection of eyes, and delirium; finally stupor, with dilatation of pupils, coma, carus, and death; no autopsy.—*Case* LX. Epidemic cerebro-spinal meningitis; stupor and lethargy were observed; next, maniacal excitement, screaming, jumping out of bed, and divergent strabismus; then coma, stertor, fits of jactitation, over-action of flexors, and spots of purpura; death by coma; autopsy.—Of these four cases three did not show any symptoms whatever of fever.—When febrile symptoms are present in this disease they are due to the local inflammation, and symptomatic of it.—Experience of Niemeyer, Ziemssen, and Githens on this point stated.—Author's conclusions as to the nature of the disease stated.—*Case* LXI. Malignant purpuric fever; febrile symptoms commencing with chill, headache, etc.; on the fourth day numerous hæmic spots appeared; they spread rapidly, and the patient died with symptoms of pulmonary obstruction in about forty hours, the febrile excitement continuing to the last; autopsy; found pulmonary and renal extravasation; skin and mucous membranes extensively blackened with effused blood, etc.—*Etiology of Epidemic Cerebro-Spinal Meningitis.*—1. Influence of age; statistical table.—2. Influence of sex.—3. Occupation and condition in life.—4. It prefers the hardy, but sometimes attacks the sickly.—*Case* LXII. Epidemic cerebro-spinal meningitis; it followed an attack of diarrhœa; sensations much disordered; death by coma; autopsy.—Two cases observed by Billroth related.—5. Influence of temperature.—6. Is the disease communicable?—7. Is it a form of typhus?—8. Is it a form of paludal fever?—The disease is due to a specific poison; it is a local phlegmasia due to a specific poison.—*Anatomical Appearances or Changes;* they result from inflammation of the cerebro-spinal meninges.—*Symptoms and Course;* these can be fully explained by the changes which the inflammatory process induces in the membranes of the brain and spinal cord; the individual symptoms are considered in detail.—*Diagnosis;* it is not attended with any phenomena which are characteristic; but the diagnosis can generally be made without difficulty; the irregular or exceptional cases are the only ones that give trouble, and they can usually be diagnosticated with certainty by exclusion.—*Prognosis;* the disease is always very dangerous; it is much more fatal in some epidemics than in others, in some parts of the same epidemics than in others, and at some periods of life than at others; each of these points is illustrated; one half of all the deaths have happened before the fifth day; the unfavorable prognostics enumerated; the favorable do.—*Treatment;* the disease is a topical inflammation which has been excited by a specific poison; the therapeutical indications enumerated; the severest cases generally prove fatal, whatever the plan of treatment may be; the remedial measures which have proved beneficial are the local abstraction of blood, the energetic application of cold to the head and spine, the judicious employment of counter-irritants, the liberal use of opium or morphia, combined in some instances with quinine, in others with calomel, but in most cases given by itself; iodide of potassium in the later stages of the disease; inutility of brandy, ammonia, camphor, valerian, musk, sulphite and bisulphite of soda; alimentation of the patient; sanitary measures which should be employed.

Synonyms.—FRENCH, *Méningite Cérébro-Spinale Epidemique, Fièvre Cerebro-Spinale, Fièvre Purpurée Maligne ;* GERMAN, *Epidemische Meningitis ;* ITALIAN, *Febbre Cerebro-Spinale.*

IT is doubtless true, as Dr. N. S. Davis remarks (vide *Transactions of the American Medical Association*, vol. xvii. 1866) and Dr. Clymer approvingly quotes, (vide *Dr. Clymer's Article on Epidemic Cerebro-Spinal Meningitis in Aitken's Science and Practice of Medicine*, 2d Am. ed. vol. ii. p. 452,) that "in regard to the disease promiscuously styled 'spotted fever' and 'cerebro-spinal meningitis,' as reported in our literature, no less than three or four diseases have been confounded together." Now, such a use of the term epidemic cerebro-spinal meningitis is obviously improper; and, inasmuch as we do not intend to describe the several diseases to which this term has been improperly applied, but only the disorder to which it properly belongs, the following account of epidemic cerebro-spinal meningitis will be found not to embrace the phenomena of some cases which have been inaccurately published as instances of that disease. But, at the same time, we shall endeavor to give a faithful and comprehensive account of the genuine cases, drawn, as far as our opportunities will permit, directly from nature.

Definition.—An acute specific disorder, commonly happening as an epidemic, general or limited, and, rarely, sporadically; caused by some unknown external influence; of sudden onset, rapid course, and great fatality; its chief symptoms, referable to the cerebro-spinal axis, are great prostration of the vital powers, severe pain in the head and along the spinal column, delirium, tetanic and sometimes clonic spasms, and cutaneous hyperæsthesia, with, in some instances, stupor, coma, and motor paralysis; attended frequently with cutaneous hæmic spots; its morbid anatomical characters being congestion and inflammation of the membranes of the brain and spinal cord, although there is reason to believe that the evidence of these changes may be wanting, even in cases of long duration. (*Clymer.*)

Considering how long this disease has been known, how widely over the earth it has been spread, how frequently it has appeared in the form of an extensive or general epidemic, and how fatal it has proved, our knowledge of it until quite recently was singularly scanty and incomplete. Dr. Tourdes, who has shown great zeal in studying the history of cerebro-spinal meningitis, thinks that it prevailed epidemically in Europe at different periods of the fourteenth, sixteenth, and seventeenth centuries. During the eighteenth century it is probable that there were outbreaks of it in France, particularly in the French fleet at Brest, (1758,) Germany, Italy, England, Ireland, and Scotland. (*Clymer.*) During the last sixty-five or seventy years it is known to have prevailed extensively in most of the States belonging to the American Union, in nearly all the countries of Europe, and in North-Africa. The first outbreak of this disease of which we have an authentic account occurred at Geneva, Switzerland, in 1805, and was made a matter of record by Dr. Vieusseux. Since that time, however, many other outbreaks of it have been described, and cases of it in great number have been published, by various observers, in both Europe and America. Within the last six or eight years, Drs. Webber, Parks, Stillé, Githens, and Clymer, in this country, and Drs. Hirsch, Niemeyer, J. Burdon Sanderson, and J. Netten Radcliffe, in Europe, have produced reports, monographs, or articles on this subject that are worthy of special mention. During this period, also, many others have presented interesting and valuable contributions on the same subject. Thus we find that our knowledge of this terrible disease is mostly of a very recent date; still, it is undeniable that this knowledge, or at least so much of it as is positive in character, is remarkably scanty and incomplete. But, at the same time, much information of value concerning it, which is negative in character, has been collected; such, for example, as that it is not a variety of typhus, nor typhoid, nor malarial fever, nor a result of poisoning, in any form, by the so-called marsh miasm; that it does not depend for its production upon any other known miasm, and that its spread is not due to contagion. Furthermore, all attempts to explain the origin of the numerous and varied epidemics of it which have from time to time appeared, or to bring them within the circle of the laws that apply to the propagation and development of diseases belonging to the zymotic class in general, have signally failed.

Nature of Epidemic Cerebro-Spinal Meningitis.—This disease is generally held, in this country and England, to belong to the great family of idiopathic fevers. Dr. Aitken defines it as "a malignant epidemic fever of an acute specific character, of sudden invasion, attended by painful contraction of the muscles of the neck and retraction of the head," etc. Hence also the terms "cerebro-spinal fever," "spotted fever," "petechial fever," "malignant purpuric fever," etc., are used as synonymous with it. Now all these different names for epidemic cerebro-spinal meningitis imply what Dr. Aitken's definition of it distinctly states, namely, that it is an idiopathic fever, and not a local phlegmasia, in its essence; and they show that the prevalent opinion concerning its nature is in favor of its being a fever *sui generis*. But is this opinion well-founded? Is it true that this disease is an idiopathic fever? This question soon forces itself upon the attention of him whose duty it is to critically investigate the clinical phenomena or symptoms of epidemic cerebro-spinal meningitis; and I am free to confess that, after much reflection on the results of my own experience and that of other observers with regard to this disease, I have been compelled to modify some of the opinions which I formerly held concerning it. It now seems clear to me, and proven beyond reasonable doubt, that epidemic cerebro-spinal meningitis is not an idiopathic fever. For, in the first place, febrile movement does not constitute an essential part of its symptomatology. It may, and often does, run its whole course without presenting any febrile symptoms whatever. Moreover, it runs its whole course in a large majority of instances without any increase at all or only a moderate rise in the temperature above the normal standard. The subjects of this disease have generally no fever-heat at all, or but little of it. In most instances, also, the pulse does not furnish much evidence of febrile excitement. In other words, the symptoms of intense fever are observed in only a small minority of the cases.

That this disorder is not *characterized* by the presence of those symptoms which constitute "fever," the history of the next three cases will show. They occurred in the author's military practice.

CASE LVII.

Epidemic Cerebro-Spinal Meningitis; at first several paroxysms of intense pain, located in the spine between the shoulder-blades, occurred; they increased in severity; then loss of consciousness, coma, and death suddenly ensued; no febrile symptoms whatever; autopsy.

Private John Minsberger, Co. I, 90th Pennsylvania Volunteers, aged 40, and sound in constitution, a patient at the Stanton U. S. Army General Hospital, Washington, D. C., convalescent from resection of the left elbow-joint, had recovered so far that the wound of operation was healed, that he had regained his flesh and strength, and that he appeared to be in good health in every respect.

On the morning of July 28th, 1864, he arose at an early hour, dressed himself, and went out to the hydrant for a drink of fresh water, as was his daily practice, and then returned to his ward, being, to all appearance, in usual health.

About 6 *o'clock* he was suddenly seized with a great pain in his back, between the superior angles of his shoulder-blades. He said it felt as if somebody was thrusting a bar of hot iron into his backbone. A mustard-plaster was applied, and he obtained speedy relief. He had no chill, and the attack appeared to begin without warning. After it was over, he got up and walked about the ward.

About 7 *o'clock* the paroxysm of burning pain in his spine returned; the sinapism was again applied, and he got relief in ten or fifteen minutes. He appeared to be sick at the stomach, and tried to vomit. His strength appeared to be good. He declined food, but had no thirst. This paroxysm was somewhat longer than the other. After it was over, he fell into a profuse perspiration, and slept an hour or two.

About 10 *o'clock* he was seized the third time with the same intense burning pain in the dorsal region, which he always referred to the backbone. This paroxysm was much severer than the others, and he cried out in his anguish. It also lasted much longer. There were no convulsive movements. The medical officer in charge of him, Acting Assistant-Surgeon W. B. Dick, U. S. Army, reported the case to me, stating that there did not seem to be much the matter with the patient during the intervals between the paroxysms, and I advised the cupping of the affected part.

At 11 *o'clock*, on raising him up in bed for the cups to be applied, he was seized with the fourth paroxysm. He fell over on the shoulder of an attendant at once, exclaiming, "Oh, such pain!" and immediately became unconscious, as if in a syncope. At the same time his face became deathly pale, his eyes fixed, his muscular system relaxed, his radial pulse weak and fluttering, and his respiration was suspended, or, rather, he breathed at very long intervals only. I was hastily summoned and in a few minutes saw him. The pallor of his face and lips had then given place to the livid hue of asphyxia; his eyes were open, fixed, and glassy, the right pupil was dilated, while the left was contracted; his muscular system was completely relaxed; no radial pulse was perceptible; and, after my arrival, he took only three or four long sighing inspirations, with long intervals between each of them, although ammonia was applied to his nostrils, and Marshall Hall's method of artificial respiration was faithfully tried. He breathed the last time about 11½ o'clock. His face and lips were then very livid. He had no thirst and no unnatural warmth of skin during any part of the five and one half hours his sickness lasted. He did not exhibit petechiæ or any other kind of spots.

Autopsy, by Assistant-Surgeon George A. Mursick, U. S. Vols., six hours after death.—Body well developed; no emaciation; no rigor mortis.

Cranium.—Upon removing the skull-cap about four ounces of blood and serum flowed out. The sinuses and veins of the brain were congested with fluid blood. There was a moderate amount of subarachnoid effusion over the hemispheres, and the ventricles contained about an ounce of serum. The spinal canal was opened from the occiput to the sacrum. The spinal cord and its membranes appeared healthy, and there was no congestion of them.

Thorax.—The lungs were intensely congested with venous blood; otherwise healthy. There were about eight ounces of serum contained in both pleural cavities. The pericardium contained about two ounces of fluid. The heart was slightly hypertrophied; the mitral valve was slightly, and the tricuspid valve was a good deal, thickened by a new deposit between the folds of the endocardium. The aortic and pulmonary valves were normal. There was no insufficiency of any of the valves.

Abdomen.—All the abdominal viscera were healthy, except the kidneys; they were in a state of acute congestion, and of a bright maroon color. The urine contained albumen; it was obtained at the autopsy.

Blood.—There was no coagulation of the blood. It remained fluid everywhere, was dark in color, and flowed freely wherever an incision was made into the body. (Vide *American Journal of the Medical Sciences*, January, 1865, pp. 21, 22.)

Comments.—The history of the foregoing case shows that epidemic cerebro-spinal meningitis sometimes destroys the consciousness and produces the other symptoms of coma with so much suddenness as to bear no inconsiderable resemblance to apoplexy, and to be liable, at first sight, to be mistaken for that disease. It therefore may, with propriety, be described in a work that is specially devoted to the consideration of apoplexy and apoplectiform disorders.

The disease in this case ran its whole course in only five and one half hours. It commenced with a sudden attack of very intense pain located in the spine between the superior angles of the scapulæ. It consisted of a series of such attacks of pain, each paroxysm, however, being decidedly stronger than that which preceded it. They were four in number, and the last one suddenly terminated in coma and death. A physician called in during the last paroxysm and finding him in a state of coma into which he had suddenly fallen, might readily have supposed the disease to be apoplexy, until informed of all the symptoms which had preceded it, that is, until he had become acquainted with the whole case. The symptoms which belonged to it were referable to disease of the membranes of the brain and spinal cord.

The rachialgia, or rather the intense and paroxysmal character of it, was a sign of some spinal lesion. It preceded the evidences of cerebral disturbance. They did not begin to show themselves until during the second paroxysm of pain, when he became sick at the stomach and tried to vomit. The nausea and retching were due to, and therefore signs of, cerebral irritation. But the disease progressed so rapidly in his head that in a few hours the functions of his brain itself were abolished, and coma with arrest of the respiratory movements, or death, ensued. Moreover, the autopsy showed precisely how this paralysis of the cerebrum and medulla oblongata had been brought about. It was by the copious effusion of serum within and upon the brain. Now, the source of this effusion of serum undoubtedly was meningeal inflammation. If the membranes of the brain in this case had been free from inflammatory disorder, the exudation of serum would not have occurred. But the effusion was poured out so rapidly as to produce almost the symptoms of "shock." The patient suddenly fell over on the shoulder of an attendant, exclaiming, "Oh, such pain!" and immediately became unconscious, as if in a syncope. At the same time a deathly pallor overspread his face. Soon, however, his countenance became livid, in consequence of pulmonary hyperæmia or stagnation, and in a short time he expired. The respiratory function failed because the respiratory movements ceased, and they ceased because the medulla oblongata and the respiratory nerves were paralyzed. Thus death began in the brain; and the meningitis which produced it ran so rapid a course that it left no marks behind which attracted attention at the autopsy, except the effusion itself.

We have treated at some length of the symptoms which were present in this case. Now, let us say a few words concerning some important signs of disease which did not show themselves in it. This man did not have a chill, and his attack occurred entirely without warning. He did not have thirst nor any abnormal warmth of the skin during any part of the time his attack lasted. He did not exhibit any sign of fever whatever. He did not have any coldness of the surface nor any other symptom which belongs to an attack of congestive fever, or to the algide stage of malarial fevers in general. We say, then, that the disease in this case surely could not have been an idiopathic fever, for it was as free from the symptoms which constitute fever as an attack of apoplexy itself.

But, perhaps, some will be inclined to doubt whether the disease in this case really was epidemic cerebro-spinal meningitis. Of such we would ask what the disease was if it was not that affection; and to such we would also remark that an undoubted outbreak of epidemic cerebro-spinal meningitis occurred at Stanton Hospital in the months of July and

August, 1864, and that the case which we have just related was the first that belonged to it.

We will now proceed to relate the second case belonging to this outbreak of the disease at present under consideration.

Case LVIII.

Epidemic Cerebro-Spinal Meningitis; first, paroxysms of extreme restlessness occurred; next, convulsive movements of an irregular character appeared; then delirium with screaming, and much tossing in bed, but without fever; dilatation of pupils, stupor, coma, and death; autopsy.

Private John Davis, Co. E, 8th Vermont Volunteers, aged 28, and of a scrofulous diathesis, was admitted to the Stanton U. S. Army General Hospital at Washington, D. C., July 26th, 1864, having an attack of intermittent fever, quotidian in type, and moderate in severity. He stated that, until recently, he had been serving in Gen. Banks's army, Department of the Gulf. Quiniæ sulph. grs. v., every six hours, was prescribed for him, and a full diet was allowed. Under this treatment he improved rapidly, and his chills ceased after the first day. The quinia was continued, and he bade fair to soon return to duty. On the morning of the fifth day after admission, however, that is, on *July* 31st, he remained in bed, complaining of loss of appetite and of feeling rather weak; his countenance expressed a good deal of anxiety, but there was no heat of skin, nor disturbance of the pulse, nor coating of the tongue. He said that he had suddenly been seized with great restlessness, about midnight, while lying in bed. It lasted about twenty minutes, but he did not sleep till toward morning. The officer of the day being called prescribed fluid extract of valerian for this attack. He had no pain. At the morning visit he was also free from pain. The quinia was continued, and he was directed to keep his bed.

He got on well through the day until *about* 5½ *o'clock* P.M., when he was again suddenly attacked with extreme restlessness, and with irregular convulsive movements, closely resembling those which occur in some cases of hysteria; he became delirious, and uttered loud, shrill screams, at the same time tossing himself about very much, and throwing himself out of bed unless restrained by the attendants; pupils dilated and symmetrical; no strabismus; pulse 100, and full, but there was no abnormal heat of skin; respirations about 14; no vomiting; could not be made to protrude his tongue, nor to swallow any thing. Ice was applied to his head and spine, a sinapism was placed on his epigastrium, and a large turpentine enema was administered; the enema was repeated in an hour, but without effect in either case. No remedy was administered by the mouth, for the power of swallowing seemed to have been lost.

At 7 o'clock P.M. the pupils continued dilated; the respirations were 12; the skin was moist; the restlessness diminished; but still he could not swallow.

At 9 o'clock P.M. pupils dilated; respirations 12, but more labored; pulse 90 and weaker; less restlessness and less screaming. Afterward the screams gradually subsided into low moans. At the same time he gradually sank into stupor and coma and carus, and died at midnight, about twenty-four hours from the beginning of the attack, and about six and a half hours after its character was fully developed. Toward the last he was bathed in perspiration.

Autopsy, by the author, ten hours after death.—Muscular system well developed; adipose tissue rather scanty; suggillations well marked in depending portions of cadaver; rigor mortis strong; scattered over the back of the neck and the dorsal aspect of the shoulders are twenty-five or thirty dark-purple spots, circular in shape, somewhat raised above the surrounding skin, and varying in diameter from that of a mustard-seed to a pea. On incising them, it is found that they present an infiltration of dark-colored blood in all the layers of the skin, and, to some extent, in the connective tissue beneath. These spots are not arranged in groups. On superficial view they present considerable resemblance to leech-bites.

Cranium.—While sawing off the skull-cap, and subsequently while removing the brain, a large quantity of serum stained with blood flowed away. It amounted in all to at least six ounces. Glandulæ Pacchioni unusually numerous, large, and adherent, for a subject of only 28 years. A moderate quantity of limpid serum beneath the visceral arachnoid membrane, especially about the vertex. The arachnoid has lost its transparency and become more or less opaque, (opacified,) especially in the same locality. A moderate amount of limpid serum in the ventricles. The choroid plexus in the fourth ventricle is thickened, pale-red in color, and presents a striking resemblance to a lamina of pale, flabby granulations, but the choroid plexus in the other ventricles presents no abnormity. The cerebrum, cerebellum, pons Varolii, and medulla oblongata were carefully examined. They were moderately congested throughout, but presented no other abnormal appearance.

Spinal canal.—After sawing through the posterior vertebral arches or laminæ on each side, and removing enough of them to open the spinal canal for almost its whole length, the theca vertebralis was seen to be distended, or, at least, well filled with serum, notwithstanding that a large quantity of cerebro-spinal fluid had already escaped while examining the brain. On laying the theca open, the spinal arachnoid membrane was found to have lost its smooth, shining appearance, and to have become opacified or pearl-colored throughout its whole extent. The subarachnoid blood-vessels were everywhere in a state of acute congestion or active hyperæmia. The cerebro-spinal fluid was noticed to contain some thin white flocculi in the lumbar region, but otherwise it was limpid and without color. The substance of the spinal cord did not present any abnormity.

Thorax.—No pleuritic adhesions; much passive hyperæmia or congestion in both lungs; an extravasation of blood, as large as a walnut, was found in the middle lobe of the right lung, at the fissure between it and the superior lobe; there was a large cicatrix at the apex of the left lung, and beneath it a vomica inclosed by a thick membranous wall, which contained a quantity of whitish, cheese-like substance. This vomica was evidently undergoing the process of contraction.[1] The heart exhibited rather more than the normal quantity of fat on its exterior, but in other respects was sound. The heart-clots were small. The blood was much more fluid than usual, and a very large quantity of it flowed into the cavity of the chest on dividing the great vessels, while examining the thoracic organs. This abnormal fluidity of the blood constituted a prominent feature of the autopsy.

Abdomen.—The liver and intestines presented no abnormity. The kidneys were intensely congested, especially about the bases and cones of the pyramids, and presented a very dark-red or almost brown color. The bladder was rather more than half full of amber-colored urine, which was found to be heavily loaded with albumen, on testing it with heat and nitric acid. (Vide *American Journal of the Medical Sciences*, January, 1865, pp. 18, 19.)

Comments.—The symptoms developed during life, and the morbid appearances revealed by the autopsy, show conclusively that this case belongs to the category of epidemic cerebro-spinal meningitis. On this point there is no room for a reasonable doubt. The clinical phenomena from first to last pointed to the brain and spinal cord, with their investing membranes, and to them alone, as the seat of the disease. The attack lasted only about twenty-four hours. It began abruptly with a severe paroxysm of restlessness or disordered sensibility, which lasted about twenty minutes. After the lapse of some hours, the paroxysm of restlessness returned with still greater severity, and it was attended with convulsive movements. Soon he became delirious, screaming, and tossing himself about very much; next, his pupils became dilated, he lost the power to swallow and to protrude his tongue, and at the same time the respiratory movements rapidly diminished in frequency; finally, stupor, coma, carus, and death occurred. The earlier symptoms denoted the existence of cerebral irritation; the later ones, that cerebral paralysis had occurred; at last the medulla oblongata became completely paralyzed, and the respiratory movements in his case ceased forever. Thus, we find again that death began at the brain in a case of cerebro-spinal meningitis.

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[1] This was a so-called tuberculous cavity in process of being cured. It was not only interesting *per se*, but also as occurring in a subject whose death was ultimately occasioned by another disorder.

The inflammatory process in the cerebro-spinal meninges of this patient was attended by a very rapid and copious exudation of serum, which was, for the most part, effused into the cavity of the arachnoid membrane. Some of it, indeed, found its way into the ventricles and into the subarachnoid connective tissue, but by far the largest part of it was effused as already stated. This serous exudation (at least six ounces of it escaped at the autopsy) was so abundant that it completely arrested the functions of the brain, including those of the medulla oblongata, by compressing the capillaries, and thus inducing anæmia of the brain-substance, that is, of the nerve-fibres and ganglion-cells which constitute that substance. Thus it happened that this patient lost the power to swallow, from the occurrence of paralysis in the muscles of his tongue and pharynx, that is, from paralysis of the glosso-pharyngeal and hypoglossal nerves; the capacity of perception, thought, volition, etc., from paralysis of his cerebrum, and the ability to breathe, from paralysis of his medulla oblongata.

The speedy death of this patient must, therefore, be directly attributed to the rapid exudation of a large quantity of serum into the cranio-vertebral cavity. Now, it is this wonderfully rapid and profuse outpouring of serum which especially distinguishes epidemic cerebro-spinal meningitis from all other forms of inflammation of the membranes of the brain and spinal cord. For example, meningeal inflammation, when produced by violence, is generally attended with an exudation of plastic lymph or of pus, accompanied, for the most part, with the effusion of a comparatively small amount of serum; and even in cases of tubercular meningitis, the effusion of serum, although it may be very copious, usually takes place much more slowly than in epidemic cerebro-spinal meningitis, and on that account life may sometimes be prolonged for an indefinite period in such cases, when the amount of the effusion happens to be very great. But epidemic cerebro-spinal meningitis is not unfrequently characterized by a very rapid and profuse exudation of serum, and in this way proves quickly fatal, as we have seen in both this and the preceding case.

Again, the disease ran its whole course in this case, as it did in the last, without producing any abnormal heat of skin, or any other symptom of fever. The clinical phenomena which it presented were all such as belong to a very grave inflammation of the cerebro-spinal meninges, and to no other disorder. The disease in this case, therefore, could not have been an idiopathic fever. Moreover, all the signs of symptomatic fever were also wanting. The foregoing cases, which followed each other in quick succession, (the former occurred on the 28th and the latter on the 31st of July,) show very clearly that epidemic cerebro-spinal meningitis may run its whole course without producing any symptom of fever whatever, and, therefore, that it is not a febrile disorder in its nature or essence.[1]

We shall next relate another case of this terrible disease, which occurred in the same hospital about a fortnight afterward. It was, however, attended with febrile symptoms of a moderate intensity.

Case LIX.

Epidemic Cerebro-Spinal Meningitis; first, debility and loss of appetite; next, nausea, thirst, feverishness, headache, and restlessness; then abnormal heat of head, injection of eyes, and delirium; finally, stupor, with dilatation of pupils, coma, carus, and death; no autopsy.

Private Simeon Bond, 37th Co. 2d Battalion Veteran Reserve Corps, aged 25, and of feeble constitution, was admitted to Ward No. 7 of the Stanton U. S. Army General Hospital, at Washington, D. C., on Sunday evening, August 14th, 1864, from the quarters of the hospital guard, to which he belonged. He had been unwell for several days, complaining of debility and loss of appetite, and the officer of the day had excused him from duty on that account. The orderly sergeant thought him to be slightly out of his head on the day before he entered the hospital, (Saturday,) and noticed that he ate nothing.

When admitted to the hospital, he was feverish, thirsty, and sick at the stomach, but did not vomit; said he had headache, and felt sick and weak; did not have chills, nor complain of any other pain; pulse about 90; the neutral mixture with aconite was prescribed. That night he was restless and slept but little.

Monday, 15*th*. He was light-headed, and did not seem to realize where he was; he wanted to get up and steal away, but was quiet withal. His head was hot, his eyes somewhat injected, his tongue furred, and his pulse 95, but stronger. Ordered the ice-bag to his head, a brisk purge, (pil. cathartic. comp. No. 3,) and spiritus Mindereri in combination with vin. antimonial. and spt. æther. nitric. A dose of morphia was administered in the evening, and he passed a better night. He had pain in the head, but not in the spine or any other part of the body.

Tuesday, 16*th*. Patient lies in a stupor, from which, however, he can be roused without much difficulty; pupils somewhat dilated and symmetrical; eyes more injected; pulse about 80, and full; respiration slower than natural, but deep and regular; he has mild delirium, and complains of headache, but sinks back into stupor again as soon as we stop troubling him; has no spasms of any kind. Ordered his hair to be cut off short emplast. cantharidis, 4 by 6 inches, to be applied to the nape of his neck, with sinapisms to his epigastrium and to the inner side of his thighs, and quinia in full doses. His bowels had been freely moved.

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[1] The pulmonary extravasation, which was found at the autopsy, also demands brief mention in the comments on this case. Its occurrence in connection with acute cerebro-spinal meningitis supports the views of Brown-Séquard on pulmonary extravasation as a result of cerebral injury and cerebral disease, already given in the chapter on pulmonary extravasation.

But he did not derive any benefit from these measures; the stupor deepened and passed into coma, in which state he died about 11 o'clock that night, something more than forty-eight hours after he entered the hospital.

No autopsy, for the body was sent away next morning at daylight, in in order to be interred at a distance, and before it could be conveniently examined. No spots on his body were noticed. The febrile movement was well marked. He did not have any convulsions. There were no symptoms whatever indicative of a spinal lesion. The symptoms all pointed to a meningo-cerebral lesion. (Vide *American Journal of the Medical Sciences*, January, 1865, pp. 23, 24.)

Comments.—This patient, like the others, died comatose. In his case, too, death began at the brain. If an autopsy had been held, we should doubtless have found profuse inflammatory œdema of the cerebral membranes, and, perhaps, of the cerebral substance also. The attack, however, did not commence abruptly. He had for several days complained of debility and want of appetite. The first objective symptom was observed by his sergeant, namely, he appeared to be somewhat out of his head. But on the day following his admission to hospital, he showed unequivocal signs of cerebral irritation. They were pain in and abnormal heat of head, injection of eyes, delirium, and restlessness or exalted sensibility. The next day after that, however, symptoms of cerebral depression, or paralysis of the cerebral functions, appeared. They were stupor, dilatation of the pupils, abnormal slowness or infrequency of the breathing, and blunted sensibility. But the stupor deepened into coma, and he died that night, like the others, of cerebro-spinal paralysis, something more than forty-eight hours after he entered the hospital.

The febrile symptoms, although distinct, were not very prominent. The heat of the skin was not at any time very great. The pulse when he entered hospital was about 90, on the next day 95, and on the day after that only 80. This febrile movement was, in all probability, symptomatic in its nature, and denoted, when interpreted in the light cast upon it by the other symptoms and the other cases, that inflammation of the membranes of his brain was his disease, and not an idiopathic fever.

The history of the following case was sent to the author, in a letter, by the late Dr. George M. McGill, U. S. Army, a very trustworthy observer. He had charge of the National U. S. Army General Hospital at Baltimore, where it occurred.

Case LX.

Epidemic Cerebro-Spinal Meningitis ; stupor and lethargy ; next, maniacal excitement, screaming, jumping out of bed, and divergent strabismus ; then, coma, stertor, jactitation by fits, over-action of flexors, and spots of purpura ; death by coma ; autopsy.

A powerful man of five feet eight inches, bilious temperament, aged circ. 30, in a stupid and lethargic condition, but still able to give his name, etc., was admitted to the National U. S. Army General Hospital, Baltimore, Md., January 14th, 1865, in company with a case of measles, and, reporting to the officer of the day that he too had measles, was sent to the proper ward with the genuine case. In this ward he became duller. Suddenly, about four hours after admission, he exhibited maniacal symptoms, crying loudly, and leaping from the bed, so that it became necessary to bind him. A careful examination of the body yielded no results, nor did the organ-interrogation as practised by the ward-surgeon. His urine was drawn by catheter—copious, but not remarkable; a little cough; pupils act with the light; slight diverging strabismus; bowels moved by enema; nose and lips dry; tongue reported dry; pulse compressible and not hard; surface moist and coolish.

Next day, quiet, semi-comatose; exhibits signs of comprehension, the ward-surgeon thinks, and appears to be sensitive of pressure in the right iliac region; breathing somewhat stertorous; jactitation by fits, over-action of flexors; urine drawn by catheter; patient resists its introduction.

Next day, purpura spots appeared over right trochanter major, and here and there; symptoms increased. From the beginning, pulse not hard, but compressible; surface moist and coolish, as stated above.

Next day he died slowly.

Autopsy.—Great muscular development and no emaciation of cadaver. Whitening of arachnoid; congestion of pia mater, and such adherence of it as lacerates the gray matter in separation; same condition in upper part of spinal marrow; choroid vein very full; pus in posterior horn of right lateral ventricle about foot of hippocampus major; none in left ventricle; lining membrane opaque; it does not glisten; serum in both lateral ventricles, and in third ventricle; no pus in third ventricle, but opacity of lining membrane; marked gelatinous tumefaction of the pineal body; marked *fibrinous* inflammation (to use a word) in fourth ventricle, and a drop of pus at nib of calamus. The whole brain-substance injected and *reddened*, cerebellum particularly so.

Intense congestion of lungs, carnification, with the most intense black congestion, accompanied with such exudative action in lowest lobe of right lung as consolidated this lobe, and made a cut portion, 1½ inches square, sink like lead in water; nodular dittos in posterior part of lower lobe of left lung.[1] Heart-clots both white and black, the latter believed to be ante-mortem, too. Heart-substance intensely congested; so also liver; gall-bladder full; spleen not overfull and rather light-colored, certainly so by comparison; intestines and kidneys full of blood, especially the former.

Comments.—Dr. McGill, in the foregoing account of his case, does not distinctly say so, but, according to my reading of it, this patient died about three days after entering the hospital. The cases of cerebro-spinal meningitis which we have related, then, present a somewhat regular gradation in respect to duration; the first survived his attack only five and one half hours, the second twenty-four hours, the third about two days, and the fourth about three days. So, too, the anatomical appearances revealed by post-mortem examination of these cases present a somewhat regular gradation; in the first, serous exudation and some congestion of the cerebral meninges were found; in the second, sero-fibrinous exudation, intense congestion, and whitening of the arachnoid; in the last, sero-fibrinous exudation, intense congestion, and purulent matter in the ventricles were found.

In this case, also, death began at the brain. Like the others, this patient died because the functions of his brain, including those of the medulla oblongata, were successively destroyed by the disease under which he labored; and it is probable that they were destroyed in about the same way as they were in the others.

Like the first two, this patient did not have any symptoms of fever, although he survived his attack for a number of days. His sickness also could not have been an idiopathic fever, nor a fever *sui generis*, nor, indeed, a fever of any other sort. We have now related, in all, four cases of epidemic cerebro-spinal meningitis occurring in soldiers, which very clearly were not instances of an idiopathic or primary form of fever. In only one of them were any febrile symptoms present, and they, in all probability, were merely symptomatic of the meningeal inflammation.

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[1] The pulmonary changes may have resulted from the cerebral lesion in the way shown by the researches and experiments on animals of Brown-Séquard, already mentioned in the chapter on pulmonary extravasation.

Moreover, the author's experience and observations on this point do not stand alone. Niemeyer, who observed the disease in Baden, remarks that prodromic symptoms are generally absent; that the disease usually commences with phenomena which point directly to the meningeal affection, and that the febrile symptoms are but trifling in comparison with the nervous phenomena. He also found that the post-mortem appearances are constantly those of an extensive inflammation of the cerebro-spinal meninges. He further notes that in his experience the temperature, on the first and second days, was often but slightly raised. (Vide *New Sydenham Soc. Retrospect*, 1865–6, p. 59.) Niemeyer also says that in epidemic cerebro-spinal meningitis, the fever and all the other symptoms depend solely on the local disease, and its injurious effects on the body, just as they do in croupous pneumonia or in erysipelas; and the changes in the meninges of the brain and spinal cord are precisely the same as those sometimes induced in other ways. (Vide *Text-Book of Practical Medicine*, vol. ii. p. 219, 1st Am. ed.) According to Ziemssen's numerous and accurate observations, the fever in cerebro-spinal meningitis has no regular course. Very few temperature curves resemble each other; sudden leaps and exacerbations of short duration often occur. But generally a remitting type, with exacerbations of half a degree to a degree, is most frequent. Very high temperatures are almost never seen except in cases that terminate fatally. In most cases the temperature does not rise at any time above 103°. (Vide *Text-Book of Practical Medicine*, vol. ii. p. 224, 1st Am. ed.) These observations certainly do not favor the hypothesis that epidemic cerebro-spinal meningitis is an idiopathic or primary fever. On the contrary, they show pretty clearly that it does not belong to that class of diseases.

Again, Dr. Githens, in his "Notes of ninety-eight cases of Epidemic Cerebro-Spinal Meningitis treated in the Philadelphia Hospital (Blockley) during the months of December, 1866, and January, February, and March, 1867," (vide *American Journal of the Medical Sciences* for July, 1867, pp. 17–39,) without aiming to elucidate the question as to the nature of this disease which we are now considering, makes the following remarks: "The *temperature* is lower than that recorded in any other typhoid or inflammatory disease. The observations were made at 6 A.M., 12 M., and 6 P.M. daily; and were taken, as far as possible, in typical cases, and in those where the temperature was most elevated, and yet the average is lower by four or five degrees than that of typhus or typhoid fever, pneumonia, etc. In two cases only did the thermometer in the axilla reach 105°. In fifteen cases it was between 104° and 105°; in twelve between 103° and 104°; in seven between 102° and 103°; in six between 101° and 102°; and in two it was below 100°; records being made in forty-four cases. ***The numbers given are the highest point reached in each case.*** The difference in the temperature at the evening and morning observations was not so marked as in most other fevers, a fall of more than one degree being unusual, and frequently there was no change. A regular and gradual descent indicated the beginning of convalescence; a rapid fall was the sure precursor of collapse." But in order to appreciate the full import of Dr. Githens's remarks, it is necessary to bear in mind that he gives the thermometric record of only forty-four cases out of the one hundred and sixty-one cases which were treated; that these forty-four cases were selected because they were considered "typical," and because the temperature in them was "most elevated," that is, was decidedly higher than it was in the other one hundred and seventeen cases; "and yet," he says, "the average is lower by four or five degrees than that of typhus or typhoid fever," etc. It is highly probable that in the other one hundred and seventeen cases, and they amount to almost three fourths of the whole number treated, the increase in temperature or febrile excitement was generally but small or moderate, and that in some it was absent altogether. Thus, the experience of Dr. Githens, when critically examined, is found to agree in substance with that of Niemeyer and the other German observers. Now, without quoting any more details from Dr. Githens's thermometric records, we will here remark that, even if his observations stood entirely alone or uncorroborated, they would throw great doubt upon the claim of epidemic cerebrospinal meningitis to be considered as an idiopathic or essential fever.

From the above-mentioned clinical and post-mortem observations made by ourselves and by several independent observers in different parts of the world, we draw the following conclusions:

1. That febrile symptoms do not necessarily belong to epidemic cerebro-spinal meningitis as a substantive disease, for it may, and not unfrequently does, occur without exhibiting any such symptoms.

2. That when fever is present in this disease it is generally but moderate in severity, and "has no regular course;" in most cases the temperature does not rise above 103° at any time; very few temperature-curves resemble each other; sudden leaps and exacerbations of short duration often occur

3. That the post-mortem appearances are constantly those of an extensive inflammation of the cerebro-spinal meninges.

4. That epidemic cerebro-spinal meningitis is by nature not an idiopathic, primary, or essential fever, but a local phlegmasia, upon which the febrile excitement, when present, and all the other symptoms solely depend.

This disease is, however, something more than a simple local phlegmasia, for it probably has its origin in an unknown special morbific agent, as we shall presently show. It is therefore a local phlegmasia of a peculiar sort or character.

We have discussed the nature of epidemic cerebro-spinal meningitis at considerable length because it seemed to us to be a topic of great practical importance. What the rational plan of treating this disease is, depends largely upon whether it is a specific fever with a more or less regular and self-limited course to run, or consists principally of a local inflammation involving the cerebro-spinal meninges. In the former case, the plan of treatment would be mainly expectant in character; in the latter case, active measures would be adopted with a view to arrest or abort the inflammatory process. We now trust that we have satisfactorily shown this disease to belong, not to the class of idiopathic fevers, but to the local phlegmasiæ, and that it essentially consists of an inflammation of the membranes of the brain and spinal cord, which is apt to be widely spread and exceedingly rapid in its progress. We have also given incidentally much of the clinical history of epidemic cerebro-spinal meningitis.

We shall next proceed to consider the etiology of this disease. But before doing so it may be advisable to relate the history of a very striking case of a disorder which might, with singular propriety, be termed "spotted" or "malignant purpuric fever," so that its clinical phenomena and post-mortem lesions may be compared with those found in the cases of epidemic cerebro-spinal meningitis which we have presented, and the differences noted. It is more than possible that such cases as the following have been improperly classified with epidemic cerebro-spinal meningitis, and that this mistake has led to much doubt, misapprehension, and confusion of ideas with regard to the subject which we have been discussing. This case was published some years ago, but soon after its occurrence, by the author, in an ephemeral journal called the *Annalist*, under the designation of "purpura hemorrhagica." The symptoms of fever, however were strongly marked and persistent.

CASE LXI.

Febrile symptoms commencing with chill, headache, etc.; on the fourth day numerous hæmic spots appeared; they rapidly spread, and she died with symptoms of pulmonary obstruction in about forty hours; autopsy; pulmonary and renal extravasation; skin and mucous membrane extensively blackened with effused blood, etc.

Catherine Tooby, aged 22, born in New-York, admitted to Bellevue Hospital, Friday, September 29th, at 10½ o'clock A.M. She says her health has usually been good; but last winter had a severe attack of rheumatism; acknowledges that her habits for some time have been licentious and intemperate.

The present attack dates from Monday, September 25th, when she took a severe chill with intense pain in the small of back and head, followed by increase of bodily temperature, thirst, and restlessness. The febrile symptoms lasted till the night of Thursday, the 28th, when she had profuse nasal hemorrhage. On the next morning (that of admission to hospital) about 7 o'clock "a breaking-out," as the patient called it, suddenly occurred. The eruption was scattered over the whole surface of her body, and its appearance was attended with great and immediate relief from pain.

September 29th, 11 A.M. Patient has been in hospital about half an hour. She presents a remarkable appearance. Her whole body is more or less discolored with purpuric spots which vary in size from a half-dime to the palm of the hand; they are most abundant on the face, neck, and chest; in some places they are almost confluent. The skin between the spots is occupied by a bright-red eruption or erythema which disappears under pressure. The conjunctivæ are dark-brown or black with echymosis; eyes watery with some defluxion from nostrils; skin hot and dry; pulse about 90 and quick; has thirst and restlessness; tongue moist and lightly coated with a whitish fur; roof of mouth also covered with a whitish coat; complains of sore throat and dysphagia; has also slight dyspnœa. Ordered acidulated drinks to be taken freely, rest, and a light but nourishing diet.

5 P.M. The purpuric spots are rather more confluent, especially on the face; skin hotter, pulse fuller; has sanious sputa; had slight epistaxis in the afternoon; dyspnœa increased. Same treatment to be continued, with the addition of tepid sponging of her body and limbs.

September 30th, 9 A.M. Patient had considerable epistaxis during night; passed two or three ounces of blood in stool; slept but very little; pulse 120; respiration 60; sputa sanious; complains of increased soreness of throat and increased difficulty of swallowing; tongue moist and covered with a brownish fur; three or four drops of sanious exudation closely resembling bloody sweat stood on her forehead and on the right side of her nose; the erythema has faded somewhat.

1 P.M. Pulse and respiration about the same as at last visit; has low delirium from which, however, she is easily roused; the bloody sweat increased; purpuric spots continue to become more confluent. Ordered acid. sulphuric. aromatic. gtts. x. secunda quaque horâ sumenda.

7 P.M. Pulse 135, small and feeble; respirations more frequent, short, and anxious; delirium still low and muttering, but now she can scarcely be roused from it; bowels have acted three or four times since morning; stools small, blackish, and very fœtid; considerable bloody sweat stands on forehead and cheeks; power of deglutition nearly gone; she has not passed any blood in the urine that was visible to the unaided eye. She continued to grow worse, and died about 10 o'clock the same evening.

Autopsy twelve hours after death.—Cadaver in good flesh and embonpoint preserved; whole surface of it blackened by the confluence of the purpuric spots and patches.

Head.—Brain and its membranes somewhat congested, but otherwise natural.

Respiratory Organs.—Larynx and trachea blackened with blood effused beneath their mucous coat. Middle third and part of lower third of each lung partially solidified by blood extravasated into the pulmonary parenchyma. These parts still contained some air. The other parts of each lung are much congested, and give out bloody fluid under pressure. Heart natural. Œsophagus and stomach completely blackened with blood effused in the submucous connective tissue. The purpuric extravasations became less abundant in the small intestines, but they increased again in size and number in the large intestines, quite down to the anus. Liver nearly double the normal size, and exhibits the fatty degeneration. Kidneys—right organ somewhat enlarged, and exhibits commencing fatty degeneration with the effusion of considerable blood into the connective tissue on its exterior; also one large and numerous small spots of extravasation beneath the mucous membrane lining its pelvis; left kidney healthy in structure, but it presents similar spots of hemorrhagic effusion. Spleen softened. Other organs healthy.

Comments.—The attack in this case was attended with the symptoms of fever, and for three days the patient seems to have presented only the phenomena of some febrile disorder. Then, however, hæmic spots in great number suddenly appeared on her skin, and she came to hospital. But the sanguineous effusion went on, nevertheless, not only beneath the skin, but beneath the mucous membranes of the respiratory, digestive, and renal apparatus, and in the pulmonary substance also; and she died of pulmonary obstruction in something less than thirty-six hours after entering the hospital. In her case, death began at the lungs. She did not exhibit any symptoms whatever that pointed to a cerebro-spinal lesion, and, at the autopsy, the brain and its membranes were found to be healthy, excepting some passive congestion which was probably due to the mode of dying. This woman appears to have had a fever, the symptoms of which lasted till her death. She was carried off by the strange hemorrhages of an interstitial or parenchymatous character, which in the end destroyed her lungs for breathing purposes; and her disease might well bear the name of "malignant purpuric fever."

In epidemic cerebro-spinal meningitis, on the other hand, the symptoms form the very first point to the lesion of the cerebro-spinal meninges, and the febrile phenomena are generally trifling in comparison to the signs of cerebro-spinal disturbance throughout the whole course of the disease. Indeed, the contrast between the symptoms which are developed in cerebro-spinal meningitis, on the one hand, and in cases such as that which was just related, on the other, is very striking and instructive. They bear some slight resemblance to each other in only two particulars, namely, hæmic spots are also sometimes present in cases of epidemic cerebro-spinal meningitis, and both are liable to quickly terminate in death. But in most other respects they are as dissimilar as scarlet fever and tetanus.

Etiology of Epidemic Cerebro-Spinal Meningitis.

1. On the influence of *age* in causing this disease. While no age is entirely exempt, from the infant a few weeks old to the octogenarian, it selects its victims principally from the young. During the Prussian epidemic of 1864–5, out of 347 deaths which were produced by it in the district of Berendt, all but 17 occurred in children under fourteen years of age. (Vide *New Sydenham Soc. Retrospect*, 1865–6, p. 56.) With regard to the ages of 161 cases treated in the Philadelphia Hospital during the winter of 1866–7, Dr. Githens remarks: "The patients ranged from 8 to 68 years of age. The character of our hospital prevents us from having many children among our patients. . . . Of the whole number scarcely one third had reached the age of forty years. One half were between the ages of fifteen and thirty." (Vide *American Journal of the Medical Sciences* for July, 1867, pp. 34, 35.) In another collection of 116 cases, wherein the ages are given, there were between 1 and 15 years inclusive, 39; between 16 and 30 years inclusive, 64; between 31 and 46 years inclusive, 12; and 1, aged 68 years. (*Webber.*) Niemeyer says: "Among the different ages, childhood has the greatest quota of cases and deaths. Persons of middle age also are often attacked, while the aged are rarely affected." (Vide *Text-Book of Practical Medicine*, vol. ii. p. 219, 1st Am. ed.) The following table explains itself and tells a very plain story.

Table showing the age, sex, etc., of those who died of cerebro-spinal meningitis in the city of New-York, during the years 1867, 1868, and 1869. Compiled from the official reports of the Metropolitan Board of Health, for those years, by the author.

		Under 5 Years.	5 to 10.	10 to 15.	15 to 20.	20 to 25.	25 to 30.	30 to 35.	35 to 40.	40 to 45.	45 to 50.	50 to 55.	55 to 60.	60 to 65.	65 to 70.	70 to 75.	75 to 80.	Total.
1867.	Males....	10	1	1	2	1	1	..	1	1	..	..	1	1	..	..	..	20
	Females..	9	..	2	..	..	1	..	..	..	..	..	..	..	..	..	..	12
1868.	Males....	12	3	..	1	..	..	1	..	1	1	..	..	..	..	..	..	19
	Females..	10	3	1	..	..	..	..	..	..	..	..	..	1	..	..	..	15
1869.	Males...	11	1	2	..	1	..	1	..	..	..	..	..	1	..	..	1	18
	Females..	17	..	1	1	2	1	..	..	1	1	..	..	..	..	..	..	24
Total...		69	8	7	4	4	3	2	1	3	2	..	1	3	..	..	1	108

Under 15 years 84; over 15 years only 24; total 108 deaths. One case was very aged, being between 75 and 80. Only 10 were more than 40 years old.

It is presumed that epidemic cerebro-spinal meningitis is meant in the health-reports referred to above and from which the table is compiled, from the fact that it is not classified under the head of the local diseases, but under that of the zymotic diseases in those reports.

2. As to the *sex* of those attacked with epidemic cerebro-spinal meningitis. Dr. Githens informs us that of the 161 cases treated in the Philadelphia Hospital during the winter of 1866–7, 122 were males and only 39 females, the ratio being rather more than three males to one female. Of the 108 fatal cases which we have tabulated, 57 were males and 51 females. This result shows that the difference between the sexes, as to liability to contract the disease, in that series of cases, was not very great. On this point Dr. Clymer justly observes: "It has been very capricious with regard to sex, in various epidemics and localities, sometimes males being chiefly its victims, at other times females." It has, however, been generally remarked that males are more prone to this disease than females.

3. On the *occupation* and *condition in life* of those who have been its victims. Vieusseux, in giving the first authentic history of an epidemic of this disease, which occurred at Geneva, in 1805, says that it attacked people of every rank at once; the poor and rich, the inhabitants of dirty and crowded rooms in unhealthy districts as well as the residents of great houses, where well-aired chambers had but a single occupant. (*Tanner.*) Dr. Githens remarks, in regard to the cases treated in the Philadelphia Hospital during the winter of 1866–7, that: "With few exceptions they were intemperate and irregular in their habits; dwelling in the most filthy portions of our city, in crowded tenements, and frequently in underground apartments, and in many cases not coming into the hospital until the chances of recovery were seriously compromised." (Vide *American Journal of the Medical Sciences* for July, 1867, p. 35.) Dr. Githens also says: "Sporadic cases occurred all through the house, being confined to no particular part, two successive cases rarely coming from the same ward." (*Ibidem*, p. 35.) Dr. John Simon writes: "Epidemics have seemed particularly apt to occur in establishments where masses of special population have been living in common domicile—as in workhouses, convict prisons, schools, and (above all) barracks. And in several such cases the epidemic has seemed to confine itself to one section of the establishment—to one block of buildings, to one floor, or to one room. It is asserted that, as a general rule, the affected segment of population has been in over-crowded and ill-ventilated quarters. And when the disease has spread from such

centres, or has independently arisen among common populations, this, almost always, has been said to have been under similar unwholesomeness of circumstances. Where the epidemic has been among soldiers, officers have enjoyed almost entire immunity; and where common populations have been suffering, the disease has shown great, if not exclusive, preference for the worst-lodged classes of the community. . . . . In some cases, according to local reports, the distribution of an epidemic has very decidedly not been governed by conditions of over-crowding and ill-ventilation." (*Clymer.*) The last statement may be illustrated by an observation of Dr. Githens: "During the winter season the sleeping-rooms in the out-wards become very crowded; and they were in this condition all of the past winter, so that wards calculated to accommodate twenty-eight persons were crowded with from seventy to ninety; mattresses being placed on the floor of the apartment, raising the person but a few inches above the floor. Notwithstanding this condition of things the epidemic did not spread." (*Op. cit.* p. 35.) In France, epidemic cerebro-spinal meningitis is looked upon as a military disease; soldiers in garrison, and particularly recruits, being the chief sufferers. In our own country, Dr. J. J. Woodward, speaking of it as it appeared in our army during the late war of the rebellion, says: "Recruits have not escaped, and those have especially suffered who were crowded in barracks and draft rendezvous." (*Clymer.*) But this disease not unfrequently prevails to an alarming extent in civil practice and in purely rural populations, where it often proves to be no respecter of persons. Indeed, it affects the villages and isolated farm-houses of the interior as much, at least, as the large cities. Although it prefers soldiers and children, no class is wholly free from its ravages.

4. While it generally selects its victims from the young, the robust, and the healthy, it sometimes attacks those who are already very ill of another disease. For example, a case is given in the First Report of the Boston City Hospital, pp. 124, 125, in which it supervened upon an attack of double pneumonia in a young Irish seamstress, aged 22. Her condition had improved until the appearance of cerebro-spinal meningitis, which proved fatal in twenty-eight hours. In the following case it supervened upon a severe attack of camp diarrhœa.

Case LXII.

Epidemic Cerebro-Spinal Meningitis; the attack followed diarrhœa; sensations much disordered; death by coma; symptoms imperfectly described; autopsy.

A soldier died rather unexpectedly of coma, in the Hospital of the 2d Division, 5th Corps, January 22d, 1865, three days after admission thereto. He had suffered from diarrhœa with thin, watery-looking discharges for about a week; was conscious till a few hours before death; had no convulsions; complained of feeling very hot, when to observers he was cold; died of coma as stated above, there being no apparent cause for it except cerebro-spinal disease.

Autopsy, four hours after death.—Cadaver in good flesh; rigor mortis strong; hypostatic congestion light.

Head.—On removing the calvarium, about half a pint of dark, almost black, bloody fluid escaped; the dura mater had been lacerated by the saw; the dura mater also presented an injected appearance. The veins on the surface of the cerebrum were highly injected. On incising the substance of the cerebrum, blood escaped in appreciable quantity from the puncta vasculosa. Lateral ventricles without fluid, but their walls were darker in color than usual; choroid plexus dingy-red. Veins of spinal cord much injected. Fluid in the spinal canal. Rather more than an ounce collected in the posterior fossa, some of it furnished, no doubt, by the spinal canal, from which more fluid (f. ʒ j. or f. ʒ ij.) came when the body was elevated.

The examination of the other organs did not reveal any cause for the cerebral symptoms, nor for the fatal result, that was situated or belonged in them.

For the notes of this case the author is indebted to Surgeon Charles Page, U. S. Army, who witnessed the autopsy.

In another case which we have related, namely, that of John Davis, (No. LVIII.,) epidemic cerebro-spinal meningitis presented itself during convalescence from an attack of malarial fever. It is, however, unquestionably true that the young and vigorous are attacked by it much more frequently than the feeble, the sickly, and the aged.

Again, it is possible that this disease may supervene in cases where important surgical operations have just been performed, and, proving quickly fatal, may be an unexpected cause of death in surgical practice. It is probable that such cases as the following, related by Dr. Billroth, of Vienna, belong to the same category as epidemic cerebro-spinal meningitis. The age of these patients, (they were both quite young,) and the circumstance that such disorders as these patients had have never before been described as the sequels of surgical operations, speak somewhat in favor of the hypothesis that they do so belong.

The *first* case was that of a boy, aged 5, who suffered from suppuration in and around the left knee-joint, the result of a fall. On March 11, 1868, Billroth opened the joint, cut and scraped away the diseased parts, and introduced drainage-tubes through abscesses which had formed in the popliteal space and under the tendon of the quadriceps extensor muscle. There was very little bleeding; no secondary hemorrhage. After the operation the child complained much of pain in the part operated on, which was partially relieved by two subcutaneous injections, each containing one eighth of a grain of morphia. On the morning after the operation, the patient having had a tolerably quiet night, the pulse was 152, the temperature 38.6°Cent. (101.7° Fahr.,) and he still had much pain. At 8 P.M. of this day—thirty-three hours after the operation—the temperature having risen to 104° Fahr., he was seized with convulsive twitchings of the lower limbs, the pain being exacerbated. After one eighth of a grain of morphia had been injected subcutaneously, he was somewhat more quiet for a time, but at half-past 10 violent convulsions, accompanied with an increase of the pain, took place in all the limbs, and even in the muscles of the trunk. One sixth of a grain of morphia was injected and produced some relief; but at midnight all the symptoms returned. Soon afterward he became comatose and died. The total quantity of morphia injected in less than forty hours did not amount to one grain. On post-mortem examination the skull was found to be thin and compact; the sutures were indistinct, and the parietal especially was closed. The dura mater was distended and its veins were full of blood; the inner membranes contained a moderate quantity. The cerebral convolutions were much flattened, the sulci were effaced; the brain was anæmic and moist, and of doughy consistence. The ventricles contained each about a drachm of clear serum. The spinal cord was softened to the consistence of pap, for a space about three fourths of an inch long, opposite the lower dorsal vertebræ; in other parts the cord was healthy. No trace of tubercle could be found in any part of the body.

The *second* case was that of a lad, aged 16, who had had a bronchocele for three years, which had grown rapidly, and extended to both sides of the neck. It caused dyspnœa, and, as it was probable that it would soon cause death by suffocation, Billroth extirpated the portion on the left side. He had, in many instances before, removed bronchocele in the same way (by ligature and excision) with success. In the course of the night following the operation, the patient was seized with violent general convulsions, and, after being comatose half an hour, died in three hours, sixteen hours after the operation. On post-mortem examination the left pupil was found rather narrower than the right. The skull was thick and firm. The right half of the frontal suture was almost obliterated externally, and was indistinct on the inner surface. The dura mater was tightly stretched; the inner membranes contained a moderate amount of blood. The cerebral convolutions were much flattened out. The brain was moderately rich in blood, and was soft and moist; its ventricles, the lining membrane of which was thickened, contained more than an ounce of flocculent serum. No large vessels or nerves in the neck had been injured in the operation. The trachea was much narrowed. There was emphysema of the lungs and remains of antecedent pericarditis and peritonitis. The heart was healthy. (Vide *New Sydenham Soc. Retrospect* for 1869–70, pp. 226, 227.)

Each of these patients died of coma after convulsions. Each of them had acute meningitis with acute œdema of the cerebral substance. In one of them softening of the spinal cord was also present. The epidemic which commenced in Silesia in 1863, and spreading thence extended itself over Germany, is said to have reached Vienna in 1865. It is not improbable that it lingered there as an endemic disease for some time afterward. (Vide *New Sydenham Soc. Retrospect*, 1865–7, pp. 55, 56.)

5. On the influence exerted by the *temperature* and by the *season of the year* in producing epidemic cerebro-spinal meningitis. The epidemic at Philadelphia, to which we have so often referred, occurred in the winter. The outbreak at Stanton U. S. Army General Hospital, which we have mentioned, occurred in the summer. But most of the outbreaks in this country have taken place during the winter and early spring. Niemeyer says: "Epidemics of this disease are more frequent in winter than in summer, and usually disappear as warm weather begins. But there are exceptions to this, which contrast very remarkably with most epidemic diseases." (Vide *Text-Book of Practical Medicine*, vol. ii. p. 219, 1st Am. ed.) Of 182 European epidemics, 24 were in October and November, 46 in December and January, 48 in February and March, 30 in April and May, 24 in June and July, and 10 in August and September. In Sweden, of 417 local outbreaks, 311 were in winter, and 106 in summer. Of 85 epidemics in Europe and the United States, noted by Hirsch, 33 prevailed in winter, 24 in winter and spring, 11 in spring, 1 in spring and summer, 2 in summer, 1 in summer and autumn, 1 in autumn, 1 in autumn and winter, 3 in autumn, winter, and spring, and 6 throughout the whole year. (*Clymer.*) The foregoing statements and figures show very clearly that outbreaks of this disease occur much oftener in winter and spring than in the other seasons of the year, and support the idea that depression of, or perhaps sudden changes in, the temperature, may exert considerable influence in producing it. Burdon-Sanderson thinks that severe cold is at least a very powerful predisposing cause; and, with Tanner, we must allow that the development of the disease is favored by cold.

6. *Is epidemic cerebro-spinal meningitis communicable from one person to another?* The brief outbreak which we witnessed at Stanton

Hospital in the summer of 1864 did not exhibit any evidence whatever of personal communicability. Dr. Githens says "it showed no signs of contagiousness" at the Philadelphia Hospital during the winter of 1866–7. The members of the Massachusetts Medical Society were very generally agreed as to its non-infectious character, and report that out of 268 cases in which the question was asked as to the origin through contagion, the replies were in the negative, so far as 252 were concerned. (*Tanner.*) Besides, there is one fact which emerges from the extensive histories of the late German epidemic with great uniformity, namely, the non-existence of any thing like well-marked contagiousness in epidemic cerebro-spinal meningitis. This is particularly noted by Sanderson, and the same story is told by all the principal German observers. (Vide *New Sydenham Soc. Retrospect*, 1865–6, p. 61.) Thus the mass of testimony is against its being contagious; but Drs. Hirsch and Stokes have reported cases of apparent communication of the disease from the sick to the well; and Boudin gives instances of its appearance in garrisons, and among the civil population of towns, after the introduction of troops among whom the disorder had prevailed or was prevailing at the time. (*Clymer.*) Now, while we cannot assert that there is no proof whatever of the personal contagiousness of this disease, we can safely say that there is comparatively little of such proof, also that it is but slightly if at all infectious, and that it certainly is not communicable from one person to another in the same sense that typhus and typhoid fevers are.

7. *Is this disease a form of typhus?* Niemeyer says: "The consideration of this disease as a peculiar form of typhus, which was formerly so common in France, has been entirely disproved during the late epidemic in Germany. I separate this affection from the class of infectious diseases to which the different forms of typhus belong, on the following grounds: In the latter, the severe constitutional symptoms, especially the fever, for the most part, depend immediately on the reception of the infecting material into the blood, and the anatomical changes in the organs, caused by the infection, are very peculiar; they are induced only by infection with the specific poison. In epidemic cerebro-spinal meningitis, on the contrary, the fever and all other symptoms depend solely on the local disease induced by the infection and on its injurious effect on the body, just as they do in croupous pneumonia or in erysipelas; and the changes in the meninges of the brain and spine are just the same as those sometimes induced in other ways." (Vide *Text-Book of Practical Medicine*, vol. ii. p. 219, 1st Am. ed.) Dr. Burdon-Sanderson says that the facts observed at Dantzig afford no proof of there being any thing in common between epidemic cerebro-spinal meningitis and typhus, except so far as each was due to a specific

poison. (*Clymer.*) Dr. Luther Parks writes: "We can conceive that on the negative side of the question of the connection of 'spotted fever' with typhus the same line of argument may be used as that of Dr. Holmes in speaking of the epidemic of 1806 to 1815—that a disease which is sometimes almost as sudden in its invasion as a stroke of lightning; which is rarely suspected of being contagious; which gives us a solitary case in a ship-of-war, a single case in a boarding-school, two cases only in an almshouse; which in civil practice affects the villages and isolated farm-houses of the interior (where typhus 'running the ordinary course' is unknown) as much, at least, as the large cities; which, in a great majority of cases, is fatal in a few days or even hours; the mortality of which is very variable; such a disease presents so many points of difference, when compared with British typhus, that we should hesitate before pronouncing the two identical." Dr. J. Netten Radcliffe says: "It differs from typhus in the aspect of the patient, rhythmical progress, range and course of temperature, form of cerebral affection, character of eruption, sequelæ, rate of mortality, anatomical lesions, and manner of dissemination. Differing in all essential particulars, doubt can only arise when the two diseases prevail together." (*Clymer.*) Besides, we have elsewhere shown that it is not contagious in the same sense as typhus is, and finally that it does not belong to the family of idiopathic or essential fevers.

8. *Is epidemic cerebro-spinal meningitis a form of paludal fever?* It has by some been regarded as a form of pernicious paludal fever, but, as Dr. Clymer justly observes, there are no sufficient grounds to believe it to be of malarious origin. On the contrary, there is good reason to believe it to be not of malarious origin, for it has been found to prevail alike on the uplands and on the lowlands, in districts which are entirely free from the typical forms of malarial disease, as well as in those where malarial disorders, such as intermittent and remittent fevers, abound. We have also shown that the symptoms of fever are not a necessary element in the symptomatology of epidemic cerebro-spinal meningitis, and that it is essentially a non-febrile disease.

But it is something more than a simple local phlegmasia, for it has a distinctly epidemic character, although it sometimes occurs sporadically. The author saw such a case at Exeter, N. Y., in consultation with Dr. David W. Patrick, in the summer of 1860. It occurred in the person of a robust farm-hand, and proved quickly fatal. Nobody else was affected.[1]

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[1] The author saw another isolated case of cerebro-spinal meningitis at Richfield, N. Y. The subject was a young girl, and she died after several days' illness. Can the sporadic cases be distinguished from the epidemic cases of this disease by any difference in the *clinical phenomena* and *post-mortem* lesions? The answer is, that they can not be so distinguished, and that in

Many others are upon record. One of the most interesting is that of a newly-enlisted man in the Mississippi Squadron in 1864—the only one—in which the meningeal lesions were well marked. Dr. Gilbert has reported two sporadic cases which he saw at Gettysburg, Pa., in 1844, and another in Philadelphia, in 1846. Dr. Clymer saw a single case in one of the U. S. Army General Hospitals at Beaufort, S. C., in the winter of 1863–4, and several more in the Army Hospitals at Savannah, Georgia, in the spring of 1865. Dr. Samuel Wilks saw one case in each of the years 1856, 1858, 1859, in London; Dr. Day, a fatal case at Stafford, in 1859, and another in 1865. (*Clymer.*) But this circumstance does not destroy its claim to be ranked among the diseases which are undoubtedly epidemic in character, for several hundred outbreaks of it have been recorded. It has, however, some peculiarities as an epidemic, which Dr. Clymer has pointed out; for example, it may, and not unfrequently has, appeared as an endemic or epidemic, limited to one place or district, or even institution or family; and again, notwithstanding its wide geographical distribution, there is no corresponding diffusion of it among the population where it prevails, for, as a rule, it is limited during an outbreak to certain localities and to certain portions of the population of such localities. The distribution of the disorder seems to be by a series of isolated eruptions rather than by general spreading.

But none of the causes which we have enumerated appears capable of accounting for the occurrence of epidemic cerebro-spinal meningitis. Those of them which seem to exert the most influence on its production, such, for example, as a low temperature, a military occupation, and a youthful period of life, are at best only predisposing causes for its appearance. Moreover, we are not acquainted with any other cause to which its origin and spread can be referred. The idea that either over-crowding, want of ventilation, badness of food, or other sanitary defects have any very direct or important share in producing it, is rejected by Burdon-Sanderson and by the best German authorities. (Vide *New Sydenham Soc. Retrospect*, 1865–6, p. 61.) These, too, are at most only predisposing causes. Thus we are led by the process of exclusion to see that the production of epidemic cerebro-spinal meningitis cannot be attributed to any single known contagion, miasm, or sanitary defect, nor to any atmospheric or telluric influence with which we are acquainted; and we are also led to believe it probable that its origin and diffusion are due to the presence of a special morbific agent or a specific poison. We therefore hold that this disease is not a simple local phlegmasia; it is a local phlegmasia which is produced by the action on the organism of a specific poison. It is well

these respects they present precisely the same features. Practically, these are all cases of the same disease, known that certain deleterious agents, when present in the blood, show a remarkable preference for particular tissues and structures of the body, and, as a result of this preference or affinity, kindle or excite in them the inflammatory process. Familiar illustrations of this statement are afforded by the effects which the gout-poison produces in the small joints of the extremities, by the changes which the rheumatism-poison induces in the large joints, endocardium, pericardium, and pleura, and by the morbid action which the erysipelas-poison excites in the skin. It is not improbable that the specific poison on which epidemic cerebro-spinal meningitis depends for its existence enters the blood, and acts upon the cerebro-spinal meninges in a similar manner. But with regard to this injurious agent or specific poison itself, by whose action this form of meningeal inflammation is lighted up, and by whose spread over greater or less extents of country more or less extensive epidemics of this disease are produced, we are entirely in the dark. What it is, constitutes the problem in the etiology of this affection, which now remains for the pathologist to solve. It has, indeed, been compared to the active factors of influenza, typhoid pneumonia, and diphtheria, by some physicians, but they agree with each other in nothing, except the obscurity of their nature. No light is thrown upon the subject by this proceeding.

Since the foregoing was written, epidemic cerebro-spinal meningitis has raged in the city of New-York with considerable fury. The first case was reported to the Health Department, February 7th, 1872. Between that date and November 14th, 1872, 939 cases were reported to the health authorities.[1] For these figures and dates I am indebted to *Dr. E. H. Janes*, the present City Sanitary Inspector. Cerebro-spinal meningitis had, however, been endemic in New-York for several years. The Annual Reports of the Board of Health show that 32 deaths from this disease occurred in 1867; 34 in 1868; 42 in 1869; 32 in 1870; and 48 in 1871. As the winter of 1871–2 advanced, the number of deaths from this cause increased with considerable rapidity. Between January 13th and March 17th, 31 fatal cases were reported. At the last-named date the mortality from this disease had become so great that it was deemed proper to give it a special column in the printed weekly statement of mortality issued by *Dr. Charles P. Russel*, the Registrar of Records, to whom I am indebted for the following figures, which illustrate the course of the epidemic better than many words of description:

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[1] It is probable, however, that these figures do not embrace all the cases which occurred during that time, for in some instances the physicians may have failed to make a correct diagnosis, and in other instances, where recovery ensued, they may have neglected to make any report at all. It will therefore be impossible to determine the ratio of mortality which has attended this epidemic with any considerable degree of accuracy; but it was doubtless more than 50 per cent.

Table showing the number of deaths from cerebro-spinal meningitis, in the city of New-York, which were reported weekly on the following dates:

		Brought forward	409	*Brought forward*	630
1872. March 23	12	1872. June 8	38	1872. August 24	6
" 30	26	" 15	32	" 31	4
April 6	39	" 22	28	September 7	5
" 13	49	" 29	23	" 14	7
" 20	30	July 6	24	" 21	4
" 27	39	" 13	20	" 28	9
May 4	44	" 20	17	October 5	2
" 11	50	" 27	10	" 12	9
" 18	51	August 3	8	" 19	3
" 25	33	" 10	12	" 26	9
June 1	36	" 17	9	November 2	3
	409		630	Total	691

From the above statement it appears that the disease assumed an epidemic character early in February, that it gradually increased in frequency until the advent of spring, that it continued to rage with much fury until the beginning of summer, and that afterward it slowly declined.

It will be useful here to state what the causes of this outbreak appear to have been. First of all, we must remark that the winter of 1871–2, especially the latter part of it, was unusually severe. The temperature in February and March ranged uncommonly low; and the spring was very backward in making its appearance. The extreme and protracted coldness of the winter probably exerted an important influence in producing the outbreak.

Again, *Dr. Clymer* has called attention to the fact that the districts wherein the original water-courses had been dammed up by the filling in for streets and avenues—the districts wherein the ground was most thoroughly saturated with moisture, constituted the chief nests of the epidemic. Most of the cases were grouped on and about the made land. Extreme humidity of dwellings, therefore, appears to have been another cause of the outbreak.

Finally, *Dr. Moreau Morris*, the late City Sanitary Inspector, has pointed out some other causes which possess great importance. He says: "Wherever we have carefully examined the local conditions, it has been found that the drainage of the premises has been faulty, or that the immediate surroundings have presented such conditions as must necessarily give rise to some form of disease; cellars containing decomposed or decomposing vegetables, garbage, or other filth, in a putrefactive condition, and privy-vaults located beneath sleeping-rooms, windows in cul-de-sacs, where there were no free currents of air. The most usual defects discovered were connected with house-drainage. These cases are not confined to the abodes of the dirty, squalid, and poor, but houses of a better class, with brown-stone fronts, have furnished their victims. There can be no doubt that over-crowding, with its attendant evils, accumulation of ordure, refuse, and various kinds of filth, absence of a proper supply of pure, fresh air, and personal neglect, invite and aggravate certain epidemic tendencies; and consequently we find, on examination of the map indicating the localities where cerebro-spinal meningitis has prevailed, that the largest proportion is to be found where these conditions obtain. In wards where the population is very dense, and living under these unfavorable influences, there have been many more cases than in other wards with better hygienic surroundings." (Vide *Dr. Clymer's Monograph on Epidemic Cerebro-Spinal Meningitis*, pp. 46, 47, Philadelphia, 1872.)

Dr. Morris also stated to the New-York Academy of Medicine, May 2d, 1872, that the first series of deaths occurred in a family of eight persons residing on Eleventh avenue, of which six were children from 1 to 13 years of age, and two were adults. Four of the children died in Eleventh avenue, and one more after its removal to another house. In the frame dwelling where the disease originated, the first floor was divided up as follows: the front room was used as a tin-shop, the rear apartment was the family-room, and between these rooms the parents and some of the children slept. A potter's clay sewer-pipe led from the privy-vault through the cellar, and was defective. Foul gases escaped from this conductor, and were diffused into a closet where the children were in the habit of playing. The smell, the parents said, was like rotten dead animals. (Vide *The* (N. Y.) *Medical Record*, June 15th, 1872, p. 245.)

There is no evidence that the disease was propagated by contagion or infection. Persons ill of it were frequently removed to other localities, and yet careful investigation shows no case following exposure in individuals who were in direct contact with the sick. (*Clymer.*)

From the foregoing account it appears that several factors coöperated in producing the recent outbreak of epidemic cerebro-spinal meningitis in New-York. They were 1st, lowness of temperature; 2d, humidity of the ground and atmosphere; 3d, foul sewer-gases and imperfect house-drainage; 4th, over-crowding and various evils connected with it; and 5th, want of adequate house-ventilation.

Anatomical Appearances or Changes.—The post-mortem examination of cases which prove fatal in two or three days is generally attended with the following results: the cadaver shows no emaciation, nor protracted rigor mortis, nor extensive hypostasis; groups of dried herpes vesicles are often found on the face and other parts of the body; sometimes hæmic or purpuric spots also are found; the muscles are usually dark-red, but in rare instances they are pale. The walls of the cranium (diploë) contain much blood; the superior longitudinal sinus is distended with fluid or softly-coagulated blood; the dura mater is more or less tense, and occasionally covered with small hemorrhagic or pachymeningitic deposits. Between the dura mater and arachnoid, that is, in the arachnoid cavity, there is usually no effusion, but in the subarachnoid space an exudation is found which, in respect to quantity and quality, occupies about a medium position between the cream-like purulent exudation belonging to meningitis of the convexity, and the thin flaky puro-serous exudation of basilar meningitis. In almost all cases the convexity of the cerebrum and the base of the brain are simultaneously affected with this form of meningitis, but in the latter situation it is usually more severe. The exudation is apt to be especially plentiful about the optic chiasm, in the fossa of Sylvius, at the base of the cerebellum, and in the fissures of the cerebrum. The nerves at the base of the brain often are completely embedded in it. The substance of the brain itself is more or less hyperæmic, almost always less resistant than usual, and, in the vicinity of the ventricles, may even become pulpy. The ventricles generally contain small quantities of purulent matter; in occasional instances, however, they are distended with clear serum. (*Niemeyer.*)

The spinal dura mater is more or less hyperæmic, and occasionally very tense, especially at the lower part. Between the spinal dura mater and arachnoid, a clear or purulent opaque liquid is rarely found. The arachnoid itself usually presents no peculiarity except a decided opacity. There is more or less purulent fluid between it and the pia mater. The tissue of the pia mater is infiltrated with sero-purulent exudation. This infiltration extends very irregularly; in most cases the cervical portion is free; the process first appears in the dorsal region, thence extends to the cauda equina, and is almost exclusively on the posterior surface. The purulently-infiltrated spots form irregular humps, broader in the middle, smaller at the ends, which are usually connected together by small striæ accompanying the larger vessels. Even where the pia mater does not contain this puro-serous infiltration, its tissue appears thickened and cloudy. The spinal cord itself is more or less hyperæmic, and occasionally it is infiltrated and relaxed or softened. In a case related by Ziemssen, the central canal was dilated and filled with purulent fluid. (*Niemeyer.*)

But in the frightful cases of epidemic cerebro-spinal meningitis which prove fatal in a few hours instead of days, and to which the terms *blasting*, *fulminant*, or *siderant* have been applied, it is not unfrequently found that the inflammatory changes in the cerebro-spinal meninges have not progressed so far, and are not so strongly marked, as those described above. For example, in a case which we have related, where life was destroyed in the short space of five and a half hours from the beginning of the attack, (see Case LVII.,) no lymph nor purulent matter was found.

The veins and sinuses of the brain, however, were congested with fluid blood, although a large quantity of bloody serum had escaped on opening the cranium. There was a moderate amount of subarachnoid effusion over the hemispheres, as well as at the base of the brain, and the ventricles contained about an ounce of clear liquid. The visceral arachnoid was more or less cloudy and opacified. In another fulminant case, where life was protracted for some hours longer, no purulent matter was discovered, but some fibrin in the shape of thin white flocculi was found in the cerebro-spinal fluid of the lumbar region. (See Case LVIII.) A very large quantity (at least six ounces) of blood-stained serum flowed out from the cranial cavity. The membranes and the substance of the brain were hyperæmic throughout. The arachnoid had lost its lustre and become more or less opaque, or opacified, to considerable extent. A moderate quantity of limpid exudation was found in the subarachnoid space and in the ventricles; and the choroid plexus showed some evidences of thickening. The spinal dura mater was found to be distended with effusion. The spinal arachnoid had lost its smooth shining appearance, and was more or less opacified or pearl-colored throughout its whole extent. The spinal pia mater was everywhere in a state of active hyperæmia, and some flocculi of lymph were found, as mentioned above. Moreover, the blood remained fluid in all parts of the body in both of these cases, as it is apt to do when death has been suddenly produced by other causes. In a subject who survived his attack about three days, (see Case LX.,) the examination post-mortem showed intense congestion of the membranes and substance of the whole brain, whitening of the arachnoid, gelatinous effusion beneath the arachnoid, purulent and sero-purulent exudation in the ventricles, gelatinous tumefaction of the pineal body, and morbid appearances, similar to those just mentioned, in the spinal canal.

With regard to the composition of the gelatinous exudation which occurs beneath the arachnoid, etc., in cases of epidemic cerebro-spinal meningitis, Dr. Burdon-Sanderson found, on microscopic examination, it to consist of cell-like bodies, either adhering to each other so closely that they could not be completely separated, or embedded in a transparent interstitial substance, while the sero-purulent fluid which occupied the spinal subarachnoid space, and in some cases the ventricles, exhibited corpuscles and granules floating freely. The cell-like bodies, although in general resembling pus-corpuscles, did not present that uniformity of size and character which are met with in normal pus. They were usually, but not always, of regular circular contour, and varied in diameter from $\frac{1}{3500}$th to $\frac{1}{1200}$th of an inch. Occasionally they exhibited the appearance of an external cell-membrane, but in most instances this could not be made out, even in perfectly fresh exudations—cases that were examined as early as eight hours after death. They invariably contained numerous granules, some of which were cleared away on the addition of acetic acid. Those which remained were highly refractive, but did not assume any special form of arrangement. The interstitial substance was beset with granules, some of which were albuminous, and others fatty. It was most abundant and distinct on the surface of the spinal arachnoid, where it infiltrated the fine connective tissue and minute blood-vessels of the pia mater. (*Clymer.*)

The post-mortem appearances, in protracted cases, are known only from a very few observations. In one case of this sort, Niemeyer found the exudation thickened, and partly affected with caseous metamorphosis, together with considerable fluid in the ventricles. The same appearances have been noted by other observers.

So far as the other organs are concerned, such, for example, as those of the thorax and abdomen, there are no particular anomalies in cases of epidemic cerebro-spinal meningitis, except some accidental complications ; and here we should especially remark that in such cases the spleen is almost always normal. (*Niemeyer.*) We may also state that it was not found to be diseased in any of the cases which we have related.

Should the fluid state of the blood which was observed at the autopsy in Cases LVII. and LVIII. be regarded as an evidence of blood-poisoning ? In framing an answer to this question we should bear in mind that the death of each of these patients was quite sudden, and that when death is suddenly produced from the operation of other causes, the post-mortem blood is generally found to be free from coagula. Moreover, heart-clots both white and black were found in Case LX., in abundance ; that is, in a case where the patient was destroyed, not suddenly, but about three days after the attack. There is no reason, then, to believe that the absence of blood-clots, which is so often observed in the fulminant cases of epidemic cerebro-spinal meningitis, is due to blood-poisoning, for they are equally absent in other instances of sudden death, and are generally present in the cases of this disease which last long enough for the blood to acquire an inflammatory or hyperinotic condition.

The renal congestion and albuminuria which were noted in Cases LVII. and LVIII. must also be looked upon as accidental complications, for they are found in only a part of the cases affected with this disease, and likewise are met with in other acute phlegmasiæ, such, for example, as pneumonia, erysipelas, etc.

Finally, the pulmonary extravasation which was found in Case LVIII., (58) and the pulmonary consolidation which was noted in Case LX., (60) probably had their origin in the cerebral lesion, and especially in the irritation of the base of the brain, which resulted in various ways from the inflammation of the meninges. Dr. Brown-Séquard has shown by experiments on animals that mechanical injury, by cutting or crushing, of the pons Varolii, is almost always, and of other parts of the base of the brain is sometimes, attended with the extravasation of blood into the pulmonary texture. (See also what is said on this subject in the Chapter on pulmonary extravasation.) He has also shown that pulmonary extravasation is not the only immediate effect which can be observed after an irritation of the base of the brain by crushing or cutting; for anæmia, œdema, and emphysema of the pulmonary tissue can likewise be produced in that way. He furthermore says: "When I publish the details of my experiments on the influence of injuries to the brain on the lungs, I will show that, in man, diseases of or injuries to the brain very frequently produce organic alterations in the lungs. I will content myself here, to prove the frequency of this morbid influence of the brain on the pulmonary organs, to state that, out of one hundred and eighty-eight cases of organic diseases of the brain recorded by Calmeil, there was a morbid state of the lungs, especially inflammation, in more than sixty cases—that is, in one case out of three." (Vide *American Journal of the Medical Sciences* for April, 1871, pp. 561, 562; also the *Lancet* for January 7th, 1871.) He holds that the morbid influence of the brain on the pulmonary organs is not exerted through the pneumogastric but through the sympathetic nerve. Now, in view of what Dr. Brown-Séquard's experiments have shown, we think it probable that the pulmonary extravasation and the pulmonary consolidation mentioned above had their origin in the cerebral lesion, for in Case LVIII. the power of protruding the tongue and of swallowing was lost at an early period, which shows that the hypoglossal, pneumogastric, and glosso-pharyngeal nerves were paralyzed, and consequently that the parts at the base of the brain were strongly affected by the meningeal inflammation; and it is not improbable that in Case LX., also, the base of the brain was considerably irritated and otherwise disturbed by the meningeal inflammation and its products.

With regard to the anatomical lesions which were present in the epidemic of cerebro-spinal meningitis that raged in New-York, in 1872, *Prof. Clark* stated to the Academy of Medicine, May 2d, that he had always found serous effusion under the arachnoid membrane; and that fibrinous or purulent effusion had been noticed more in this disease than in other inflammatory diseases of the brain. (Vide *The Medical Record*, June 15th, 1872, p. 246.) Extended inquiry, however, shows that the *post-mortem* lesions were identical with those described above as found in other epidemics; and that in some instances the cerebral meninges, and in others the spinal, were chiefly or only affected.

Symptoms and Course.—We fully concur in Niemeyer's view that the symptoms and course of epidemic cerebro-spinal meningitis can be fully explained by the changes which are produced by the inflammatory process in the membranes of the brain and spinal cord, and that, in this respect, the malady differs from most other diseases which result from infection or the action on the system of a specific poison. Every thing that has been advanced in opposition to this view may be refuted by the simple fact that, in genuine croupous pneumonia, which no one classes among the infectious diseases, certain symptoms, such as the frequent occurrence of herpetic eruptions, albuminuria, etc., are just as difficult to explain, from the inflammatory changes in the lungs and the copious exudation in their alveoli, as are some of the *occasional* symptoms or complications of epidemic cerebro-spinal meningitis.

The attack generally begins suddenly. Only in rare instances is the outbreak of the disease preceded by a premonitory stage characterized by slight headache and pain in the back. Dr. Burdon-Sanderson examined fifty-six cases in the Vistula-epidemic of 1865, and reports that in all, except two, the onset of the disease was sudden, the patient having been in good health up to the moment of attack. This agrees with what the German observers generally relate. (Vide *New Sydenham Soc. Retrospect*, 1865–6, p. 56.) Usually the scene opens with an unexpected chill of variable duration, which is soon accompanied by severe headache, and, in most cases, by vomiting. The headache rapidly becomes very severe, the patient grows very restless, tosses about constantly, the pupils are contracted, the intellect remains clear. The pulse is from 80 to 100, the bodily temperature moderate, the respirations increased to 30 or 40 per minute. At the end of the first or second day, sometimes later, we notice that the head is drawn backward. At this time there is often a herpetic eruption about the mouth, on the cheeks, eyelids, ears, and occasionally on the extremities. The complaints about severe headache continue; the pain extends from the head to the nape of the neck and the back. The restlessness becomes excessive, the ideas confused, the pupils remain contracted, the belly is sunken, and the bowels are constipated. The pulse and respiration become more frequent, occasionally the pulse is over 120, and the respiration over 40 per minute; the bodily temperature still remains proportionately low; sometimes, however, it rises to 103° or over. On the third or fourth day of the disease the tetanic contractions of the muscles of the neck and back become more evident, and are occasionally accompanied by trismus; there is excessive opisthotonos; consciousness is lost, but the patient still tosses about in bed; the pupils remain contracted, constipation continues, the belly is still sunken, urine is passed involuntarily, or else the bladder becomes distended, and must be evacuated by the catheter. The now unconscious patient falls into deep stupor, the moaning respiration is accompanied by moist râles, and death occurs with the symptoms of acute œdema of the lungs. But, in some cases that are particularly severe, the symptoms mentioned above are far more rapidly developed; consciousness is lost during even the first day, and, at the same time, the head is drawn far backward by severe tetanic spasms of the muscles of the neck and back. Death may likewise occur on the first or second day when the disease is very malignant. Lastly, in some few cases, (of which there can be no doubt,) the disease runs a still more rapid course, (*méningite foudroyante*,) and causes death in a few hours from general paralysis, occasionally even without the appearance of those tetanic symptoms which usually attend this disorder. (*Niemeyer.*) The first two cases related in this Chapter were markedly fulminant and belong to the last-mentioned category. In neither of them was the head retracted. In both of them the attack was made with something like the suddenness of the thunder-stroke. In one of them the leading symptoms were intense pain in upper dorsal region, sudden coma, and speedy death. In the other, extreme restlessness, irregular or choreic convulsions, loud screaming, delirium, stupor, coma, and death. In this case the muscles of the tongue and pharynx were the first to become paralyzed, and, therefore, this patient lost the power of swallowing and likewise of protruding his tongue at a very early period.

This disease, however, does not always terminate fatally, even when it has commenced and run its course for the first few days in the manner above described. Among the favorable signs, we usually notice first that the jactitation grows less, and that the mind becomes clearer, while the complaints of pain in the head and back, and the tetanic spasms of the cervical and dorsal muscles continue, or only slightly decrease. If the improvement progresses, all symptoms of the disease may disappear in a few days, and the patient begins his tedious convalescence. Occasionally, improvement begins but does not continue, and the disease drags slowly on. In such cases convalescence may not occur for weeks. Headache, retraction of the neck, or opisthotonos continue. Paralysis of the motor and psychical functions also causes a complicated series of symptoms, and the majority of such patients finally die of gradually-increasing marasmus (*Niemeyer.*)

Next, we must mention the *intermittent course* which the disease sometimes pursues. Hirsch recognizes three varieties of it: The first occurs only in the forming stage of the disease; one or more than one attack of so-called premonitory symptoms passes away, but another follows, which is immediately succeeded by the fully-developed outbreak. In the second form a sudden remission of the symptoms appears; they grow worse again the next day, however, and occasionally this alternation occurs several times, usually with a more or less regular quotidian type. In the third form, which is far the most frequent, perfect intermissions are observed during convalescence. The after-symptoms, especially headache and stiff-neck, increase very considerably for some time in regular paroxysms, which are mostly of a quotidian type, while the patient feels quite comfortable during the interval. Niemeyer states that he had had a case of this sort. Our cases LVII. and LVIII. also pursued an intermittent course, but they belong to the first of the above-mentioned varieties. In one of them the attack began with a paroxysm of great restlessness, which was followed by a complete intermission; in the other, with a paroxysm of intense dorsal pain, followed also by a complete intermission, but it returned again and again, with constantly increasing severity, until finally the patient sank into coma, carus, and death.

Lastly, we must mention that *recrudescences* not unfrequently occur during the course of, or convalescence from, this disease. These recrudescences are often so slight as readily to escape notice, but sometimes so severe as to assume the character of *relapses;* and sometimes the relapses themselves look like the reproduction of the symptoms of invasion. In general, a case attended with relapses is destined to be tedious, but not fatal; the non-fatality, however, is not absolute. Adults are not so liable as children to recrudescence of the original symptoms; but often there are marked variations in the constitutional state and in the intensity of pain. (Vide *New Sydenham Soc. Retrospect*, 1865–6, p. 58.)

Again, the individual symptoms of epidemic cerebro-spinal meningitis demand some further consideration in this place:

1. *With regard to pain in the head, neck, back, and extremities.*—Severe headache is said to be present in almost all the cases, including even those which are called fulminant or siderant, and terminate fatally in a few hours. In both of our cases, (LVII. and LVIII.,) however, it was absent. In the cases which run the usual course, as long as the patients remain conscious they complain of headache, either spontaneously or on being questioned, and it is also probable that the restlessness, groaning, and screaming of the patients, after the intellect has become clouded, are partly due to the pain in the head. Sanderson remarks that the patient frequently utters piteous cries of pain, although he is so profoundly prostrate and indifferent to external impressions that he is incapable of replying to questions. Furthermore, Niemeyer states that, during an epidemic of this disease, there are almost always a few instances in which persons, without actually having the disease, complain of very severe headache

continuing for several days, without any other apparent cause, and that we may consider such cases as abortive forms of epidemic cerebro-spinal meningitis.[1]

Pain in the neck and back, it is said, usually begins at a very early period, that is, simultaneously with the headache, or very soon after it. In one of our cases, (LVIII.,) however, the only pain complained of was located in the upper part of the dorsal region. The pain in the neck and back which attends this disease is generally increased by pressure made on the spinous processes of the corresponding vertebræ; but, with rare exceptions, this pain becomes far greater when the patient makes voluntary movements of the spinal column, and likewise when it is subjected to passive motion. If the disease runs a protracted course, the dorsal pains and their increase by movements of the spinal column may continue for weeks.

Painful sensations in the extremities, unmistakably neuralgic in character, and caused by irritation of the posterior roots of the spinal nerves, are not constant symptoms; occasionally they occur only on bending or moving the spinal column. (*Niemeyer.*) Dr. Githens found that muscular pains occupied a prominent position among the early symptoms in the cases observed by him. They were present to a greater or less extent in almost every case where reliable answers could be elicited. Their usual seat was the posterior muscles of the legs and thighs, the erector spinæ muscles, and the posterior cervical muscles. These pains sometimes disappeared early, but in many cases they, with the stiffness, lasted until convalescence was established. Again, these pains occasionally took the form of a soreness, more or less severe, involving the whole muscular system and having its culminating point in the neck or back, and developing in some cases decided opisthotonos. (Vide *American Journal of the Medical Sciences* for July, 1867, pp. 36, 37.)

2. *Hyperæsthesia and Anæsthesia of the Skin.*—Usually for the first days of the disease, and in some cases during its whole course, the patients are very sensitive to any rough handling; their restlessness, groaning, and complaints are increased when they are turned over in bed, and occasionally when they are only percussed. At a later period we often see no notice of it taken even when the skin is greatly irritated; but in such cases, if the patients are in a state of stupor, there is also cerebral anæsthesia. Far more rarely a form of peripheral anæsthesia occurs, in which the pa-

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[1] Dr. Githens says that headache was a prominent and a characteristic symptom in the Philadelphia epidemic of 1866-7. Many of the patients adopted the same simile in describing this symptom, saying their head felt as if "bound with a hoop of iron." The headache was generally either frontal or occipital, although in some cases it was diffused and general. It was observed more frequently than any other symptom, having been present in 92 of the 98 cases recorded. (Vide *American Journal of the Medical Sciences* for July, 1867, p. 36.)

tient feels irritation of the skin very little or not at all, while, at the same time, he is quite conscious. This symptom apparently depends upon loss of excitability in the posterior roots of the spinal nerves in consequence of inflammation. (*Niemeyer.*) But the kind of cutaneous hyperæsthesia mentioned above is generally present at an earlier period, and sometimes to such a degree that slightly touching or brushing the skin with the hand brings on reflex muscular contractions. In one of our cases, (LVIII.,) much of the tossing and throwing of himself about in which the patient seemed to indulge was probably due in reality to the reflex movements which resulted from cutaneous hyperæsthesia, and the same remarks appear to be applicable to Case LX. Dr. Githens states that hyperæsthesia of the general surface existed in 38 out of 98 cases, in various degrees of intensity; in some instances it was very slight, but in others so severe as to cause the patient to exclaim when touched with the tip of the finger. (Vide *op. cit.* p. 37.)

3. *Tonic Convulsions.*—According to Niemeyer, tetanic spasms of the cervical and dorsal muscles are never absent except in some few cases of méningite foudroyante. At first, the head is but slightly retracted; later, it may form almost a right angle with the body. This position of the head and the addition of opisthotonos in the dorsal and lumbar regions render it impossible for the patient to lie on his back. When the tetanus attains a high grade it almost always affects the breathing. Occasionally it disappears shortly before death; more frequently, however, it continues more or less severe until death occurs or convalescence is established. There is, however, an important fact relating to this topic which stands out in Burdon-Sanderson's observations, namely, that the contraction of the muscles at the back of the neck was never so great in any of his cases as to deserve the name of tetanic. It appeared as if the attitude of the patient (with the trapezius contracted and the head drawn back) was merely the consequence of the severe pain in the muscles, the patient maintaining this position to ease himself; and this idea was supported by an analogous attitude obviously assumed for the purpose of relieving pain in the abdominal muscles. Sanderson also mentions that in some patients whom he saw early in the first stage, (on the day following the appearance of delirium,) there was not a trace of retraction of the neck, nor of stiffness, nor of any thing else remarkable in the muscles. (Vide *New Sydenham Soc. Retrospect,* 1865-6, p. 57.) Out of the five undoubted cases of epidemic cerebrospinal meningitis which we have related, retraction of the head was certainly absent in three, and is not mentioned as having been present in either of the other two. Dr. Githens, however, found opisthotonos in 17 out of 98 cases. It was accompanied by subsultus tendinum in only two instances.

He also found more or less contraction of the posterior cervical muscles in 28 cases, evidenced sometimes merely by its being impossible to approximate the chin to the breast. (Vide *American Journal of the Medical Sciences* for July, 1867, p. 37.)

Clonic convulsions of an epileptiform or eclampsic character but rarely occur in this disease; and this circumstance appears remarkable when we consider that the inflammatory exudation is often widely spread over the convexity of the cerebral hemispheres.

4. *Paralysis.*—Generally no paralysis is observed until the approach of death. A few cases, however, have been noted in which hemiplegia or paraplegia, and quite a number in which paralysis of the facial, oculomotor, or abducens, occurred. In one of our cases, (LVIII.,) paralysis of the muscles of the tongue and pharynx soon appeared, and the power of protruding the tongue and of swallowing was lost at an early period. It is not at all difficult to explain these paralyses; on the contrary, it is almost wonderful they are not more frequent. (*Niemeyer.*)

5. *Mental Disturbance.*—At the commencement of the disease, the intellect is almost always unclouded; the patients answer questions correctly. But they soon become ill at ease and very restless. Then questions prove annoying, and they give only short and incomplete answers. The jactitation, which is scarcely interrupted by pauses of a few minutes, and is therefore almost incessant, is very characteristic in the first stage of the disease. Subsequently, most patients have delirium of variable intensity, which finally gives place to a soporose or comatose condition. (*Niemeyer.*) Some patients are violently delirious; others wander quietly, sleep but little, and are often subject to hallucinations; and cases are met with in which the evidences of cerebral disturbance are, from first to last, confined to sleeplessness and night-wandering. (*Sanderson.*) In three, at least and probably in four, of the five cases which we have related, there was more or less delirium. In two it was maniacal, while in the third it was of a quiet wandering character. This patient was light-headed, could not sleep, and seemed not to know where he was; he wanted to get up and steal away, but at the same time he was very quiet. The screams and the loud cries which were observed in the maniacal cases were probably due in part to the delirium itself and in part to the intense pain which these patients suffered at this time. Dr. Githens notes that there was no delirium at all in 31 out of 98 cases; in some of them, however, there was a condition of morbid vigilance, with an anxious, restless expression of countenance. In a number of cases there was merely a loss of animation and interest—a mere stupidity and soporose condition. Sometimes this existed

during the day, with slight delirium at night. In 29 cases there was a quiet, talkative delirium, coming on periodically for a few hours in the evening, or lasting for a few days, and then disappearing. Only three of these patients evinced any desire to leave their beds. In nine cases the delirium was of a more active and even violent character. (Vide *American Journal of the Medical Sciences* for July, 1867, p. 36.)

6. *Disturbance in the Organs of Special Sense.*—The victims of epidemic cerebro-spinal meningitis not unfrequently become blind from a keratitis, which is probably induced by incomplete closure of the eyelids, due to paresis of the orbicularis palpebrarum, or from an exudative choroiditis and neuro-retinitis, that is probably a result of direct propagation of the purulent infiltration along the optic nerve from the cranium to the eye. Deafness is remarkably frequent, so that we are almost obliged to suppose that it has several causes, among which, however, the most important, doubtless, is the propagation of purulent infiltration along the auditory nerve to the internal ear. (*Niemeyer.*) Deafness occurred in ten cases seen by Sanderson; and he has no doubt, from the inspection of pathological specimens obtained from similar cases, that the cause of it was an inflammatory change in the middle ear.

During the epidemic of cerebro-spinal meningitis which prevailed in New-York, in 1872, *Dr. H. Knapp* saw forty-one cases of blindness or deafness due to that disease, and published an account of them, wherein he says:—"Among the forty-one patients thirty-one were deaf on both sides, eight were blind with one eye, mostly the left, one was blind with both eyes, and one was deaf on both sides and blind on one. In all cases the diagnosis of cerebro-spinal meningitis could be well established. . . . .

"The affection of the *eye* begins usually in the first weeks of the general disease with the following symptoms: circumcorneal injection, discolored iris, ragged pupil, fundus oculi dull, its details not recognizable, or the fundus yielding only a sombre red color, or appears black. Hypopyon and yellowish exudation plugging the pupil are not infrequent. The cornea in some rare cases becomes ulcerous; in others the conjunctiva and lids are œdematous and very red, the eyeball protrudes, and exceptionally bursts, suppurates, and shrinks. Ordinarily, the injection of the conjunctiva subsides, the cornea clears up, the hypopyon and exudation in the pupil disappear, and the eyeball assumes a strikingly peculiar and characteristic appearance, which I have only seen in cerebro-spinal meningitis, puerperal fever, and very seldom in typhoid and typhus fevers. The iris is dull and bulges forward like a cone; its periphery, however, is usually drawn backward. The pupil is rather narrow, ragged, and immovable. Through the transparent lens, which has advanced with the iris, a dull white surface is visible in the vitreous chamber. The eyeball is commonly softer and smaller than natural. Sight is completely and irrevocably lost. Later, the crystalline becomes cataractous. The eyeball will remain smaller and softer, squint outward, but never give rise to other inflammations, or sympathetic affection of the other eye. I supply these facts from my observations of the epidemic of cerebro-spinal meningitis which reigned in the upper valley of the Rhine eight years ago. This eye-affection has been mistaken for medullary cancer (glioma) of the retina, but may be distinguished from it by its acute development in combination with the general disease, the peculiar protrusion of the centre of the iris and its retraction at the periphery, the dull white reflex from behind the pupil, and the diminution of size and tension of the globe.

"The nature of the eye-affection is purulent choroiditis, probably metastatic. There have been other changes of the eye observed in cerebro-spinal meningitis, principally hyperæmia and inflammation in and around the optic disk; they are rare and not specifically dependent upon this form of meningitis, but on hyperæmia, exudation, and proliferation in the cranial cavity in general.

"The *ear-affection* in cerebro-spinal meningitis does not show symptoms so peculiar as the eye-affection. In the early stage hyperæmia of the middle ear is commonly present, the drumheads being dull, yellowish, the region of the handle and upper portion red, and the light spot faint, smaller, or absent. The pharynx is generally red. The tympanum is inflatable, with a rough blowing sound, after which the appearance of the membrana tympani is not essentially changed. In very rare cases only the affection rises beyond this condition of a mild catarrhal otitis media, developing into purulent inflammation of the drum, with perforation of the drumhead and otorrhœa, which ceases in one or several weeks. These symptoms on the part of the middle ear are, however, of subordinate significance when compared with those furnished by the *inner ear*. In some cases patients at first find sounds around them—for instance, the song of a canary-bird—intolerably harsh, but very soon the hearing-power will diminish, and in nearly all cases be totally and permanently destroyed. When the patients retain their consciousness impairment of hearing may be noticed as early as the second day of the disease, increasing day by day to total deafness in a week or two. In the majority of cases the deafness is only discovered when the patient awakes from his stupor; and may then be total or partial, increasing, in the latter instance, to total deafness very soon. I find, however, in my notes some cases in which hearing was still present some weeks after the disappearance of the severe symptoms of the acute disease, and was lost during the recovery. In all cases the deafness was bilateral, and, with two exceptions of faint perception of sound, complete. Among the twenty-nine cases of total deafness there was only one who seemed to give some evidence of hearing afterward. The treatment consisted in leeches behind and before the ear, blisters, tincture of iodine, ung. tart. stibiat. behind the ears, and the use of the galvanic current. I have seen no good results from this treatment, nor have I heard of a better one to substitute it. . . . . .

"The majority of the patients were under 10 years; above that age there were one of 10, 12, 13, 14, 16 years respectively, and two of 18 years. The nature of the ear-disease is in all probability a purulent inflammation of the labyrinth, by which the membranes of the inner ear are destroyed in a similar way as the membranes of the eye by the purulent choroiditis. Heller and Lucae have corroborated this by three post-mortem examinations. No disease of the middle ear could annihilate the hearing so completely that no sounds whatever, not even a tuning-fork vibrating on the cranial bones, is perceived. The deafness can not be the consequence of a destruction of the acoustic nerve within the brain, or of the centre of audition; for it would be unexplainable why the adjacent facial or other cranial nerves are not, sometimes at least, found destroyed too. An additional proof is furnished by Dr. Gruening, who electrized the greater number of these patients according to Brenner's method, and obtained the normal reaction of the acoustic nerve, a fact which excludes the destruction of the nerve and centre of audition." (Vide *The Medical Record*, Aug. 15, 1872, p. 341.)

7. *Cutaneous Eruptions.*—Among the eruptions, groups of herpes vesicles in large numbers are very often seen, and, more rarely, erythema, roseola, urticaria, petechiæ, and sudamina. The frequency of the exanthemata, and particularly the occasional symmetry of their occurrence, have led to the supposition that they might depend on irritation of the cutaneous nerves, as Bärensprung has shown to be the case in herpes zoster from neuralgia. (*Niemeyer.*) But whatever may be their cause, they are not essential features of the disease, and therefore vary considerably in different epidemics. Of the 98 cases noted by Githens in the Philadelphia epidemic of 1866–7, 36 had petechiæ; 13 mixed petechiæ and erythema; 9 erythema and urticaria; 3 indistinct petechial mottling; and 37 no eruption at all. That eruptions on the skin are not essential features of the disease is proved by the fact that they were absent in considerably more than one third of these cases, and that they vary considerably in different epidemics is shown by the circumstance that petechiæ were comparatively rare in the late German epidemic, that they were present in more than one third of the cases which Githens has recorded, and that they were nearly constant in the late Irish epidemic. They were observed in two of

the five cases related by us. In one they were noticed on the third day, and in the other they were not found until the autopsy was being made.

8. *Pyrexia.*—According to Ziemssen's numerous and accurate observations, the fever has no regular course. Very few temperature-curves resemble each other; sudden leaps and exacerbations of short duration often occur. But generally a remitting type, with exacerbations of half a degree to a degree, prevails. Very high temperatures are almost exclusively seen in severe cases that terminate fatally. In most cases, however, the temperature does not rise above 103°. The fever of intermittent type which occasionally accompanies the other symptoms during convalescence is regarded by Ziemssen as due to absorption of the inflammatory products, but he refers that which occurs during the first and second weeks to an interrupted progress of the meningitis itself. The frequency of the pulse does not correspond at all with the height of the fever; it is occasionally very great when the fever is but moderate. Abnormal slowness of the pulse is only rarely observed at the commencement of the fever. (*Niemeyer.*) The same author also remarks that, in his experience, the temperature was often but slightly raised, on the first and second days; and that it only rose as high as 104° or more, a little while before death. (Vide *New Sydenham Soc. Retrospect*, 1865–6, p. 59.) Sanderson, however, noticed the temperature as constantly elevated at all stages of the disease, seldom falling below 100°; but his observations stand alone, as they do not agree with those made by other equally competent observers. (Vide *New Sydenham Soc. Retrospect*, 1865–6, pp. 58, 59.) Githens remarks, as we have already stated, that the temperature is lower in epidemic cerebro-spinal meningitis than it is in any other typhoid or inflammatory disease. Thermometric records were kept in 44 cases at the Philadelphia Hospital selected as typical ones, or instances in which the temperature was most elevated, "and yet," he says, "the average is lower by four or five degrees than that of typhus or typhoid fever, pneumonia, etc." Githens also found that a regular and gradual descent in the temperature indicated the beginning of convalescence; a rapid fall was the sure precursor of collapse. (Vide *American Journal of the Medical Sciences* for July, 1867, p. 38.) In only one instance out of the five which we have related were the symptoms of fever observed, and in that one case they were but slight.

From the foregoing description we perceive that the symptoms of epidemic cerebro-spinal meningitis vary considerably in different cases. They appear to depend, in individual instances, upon the severity of the attack, the part of the cerebro-spinal membranes which is principally affected, and the constitution or idiosyncrasies of the patient. From reading the reports we also find that the symptoms vary considerably in different outbreaks; for example, in some the cerebral symptoms are more prominent, in others the spinal, etc. Moreover, the phenomena of the disease do not appear to be exactly the same in a military, urban, alms-house or hospital, and a rural population.

In the epidemic which raged at New-York in 1872, however, there was a remarkable uniformity in the mode of attack, symptoms, and course of the disease. In nearly all the cases the onset was abrupt in the midst of apparent health. The seizure was announced by a sensation of chilliness, and in some instances by a sharp rigor; this was followed by nausea and vomiting, and pains in the head, back, and limbs, with great debility. A convulsion was often the initial symptom in infants and young children. Stiffness and gradual contraction of the posterior cervical muscles soon appeared, causing retraction of the head, and this often extended to the dorsal muscles, causing also opisthotonos. Young subjects were sometimes attacked with a series of tetanic spasms. Intelligence was apt to be sluggish from the outset. Delirium was frequently an early symptom, and varied in degree from slight hallucinations to violent mania, alternating with or passing into stupor or deep coma. Some patients could be roused by sharp questions spoken close to the ear; in others there were intervals of consciousness. Excessive restlessness and sleeplessness were constant, and often there was moaning. In many young subjects there was continual screaming. Headache was always present. It was apt to increase toward evening, and generally was not referred to any particular region. The pupils contracted irregularly, and in some cases there was double, or some other form of perverted vision. Tinnitus and dulness of hearing were not infrequent, and occasionally there was total deafness. Taste was constantly impaired. Hyperæsthesia of the skin was common, and sometimes to a very great degree. Pain in the abdomen was complained of by many, and costiveness was the rule. There was generally no thirst, but indifference to food. The pulse-range was from 90 to 101, and that of the body-heat 99°–105° Fahr.; respiration was hurried, sometimes from the onset, and excessive prostration was a marked and constant symptom. The eruption when present varied in character. In some cases, it was composed of small petechial spots; in others it was bright and diffuse like the rash in scarlet fever; in many cases distinct rose-colored spots, of irregular shape, not disappearing on pressure, with an ecchymosed margin, were visible during the first forty-eight hours; slightly elevated papules containing a transparent fluid, with a central depression, as in the variolous pock, were occasionally noticed, as well as ecchymosed spots like blood-blisters; and there were true purpuric extravasations of more or less extent. The appearance and continuance of the eruption were during the first two or three days; it was rarely to be seen after the second or third day. In some rapidly fatal cases no eruption was visible until after death, when the whole surface would become covered with rose-colored spots on a ground of diffused purpura. The eruption was by no means constantly present. In very many well-marked cases, and where it was carefully looked for, it was absent. There is no doubt, too, that it sometimes escaped notice from the shortness of its duration. Herpetic vesicles were common. In the cases which recovered, convalescence was often tardy, strength being very slowly regained, and in many some disorder of the nervous system appeared, as paralysis and perversion of the special senses, particularly deafness. Stiffness and swelling of the joints were common. Relapses occasionally happened. (Vide *Dr. Clymer's Monograph on Epidemic Cerebro-Spinal Meningitis*, pp. 57, 58.)

On the above-mentioned topics *Professor Clark* remarked to the New-York Academy of Medicine, May 2d, 1872, that he had seen cases where the spinal symptoms were not developed, but only the cerebral. In one case of true spinal meningitis, however, the patient remained for two weeks in a state of opisthotonos, and finally recovered. Severe pains in the limbs continued for some time. In other cases the chief pain was in the head, the pain in the limbs subsiding. He remembered one fatal case where pneumonia set in after the brain-symptoms had subsided. In none of the hospital cases was there any ecchymotic eruption, but a papillary or herpetic variety was observed, with a depression in the centre resembling small-pox. (Vide *The Medical Record*, June 15th, 1872, p. 246.)

Diagnosis.—Dr. Githens observed in the Philadelphia epidemic of 1866–7, that the expression of countenance was peculiar, and might almost be said to be diagnostic. The corners of the mouth were drawn down; the eyelids widely opened; the eyes protuberant or ecstatic; the pupils contracted—sometimes only apparently so in consequence of the large surface of the eye uncovered. The patient had a restless, excited air, and seemed to watch with interest every motion and change of countenance of those around him. He seemed to be looking through instead of looking at an object, in consequence of the visual axis of the two eyes being parallel; in some cases the contraction or dilatation of the pupils in the two eyes did not correspond; occasionally, one became contracted while the other remained dilated under the stimulus of a strong light. Photophobia was present in several cases, evidenced even during coma by the rigid and persistent closure of the eyelids and by opposition to the forcible opening of the same. The conjunctivæ were almost always congested or injected, (eye pearly white in two cases only, accompanied by excessive hemorrhage;)

so much so, in one instance, as to reach the condition of chemosis. This hyperæmia of the conjunctivæ was followed in two cases by considerable purulent secretion; in others a few pus-corpuscles were occasionally observed. The lachrymation was frequently excessive. (Vide *American Journal of the Medical Sciences* for July, 1867, p. 37.)

Epidemic cerebro-spinal meningitis, however, is not attended by any single symptom nor by any one group of symptoms which characterizes it or distinguishes it from all other diseases. But at the same time the symptoms which it produces, when considered as a whole or taken into account all together, are generally so well marked that the diagnosis is made, in most cases, without difficulty. The suddenness of the attack, the rapidity with which the symptoms are developed, the presence of more or less severe pain in the head or spine or in both situations from the outset, the occurrence of phenomena denoting that the special functions of the brain and spinal cord are disturbed or suspended, and, in many cases, the peculiar character of these evidences of cerebro-spinal disturbance—such, for example, as retraction of the head, extreme restlessness, etc.—together with the absence of the symptoms which characterize other disorders that bear some resemblance to it, generally render the diagnosis both easy and certain. Difficulty in differentiating epidemic cerebro-spinal meningitis from other disorders is experienced only in exceptional instances; and these not unfrequently belong to the class denominated fulminant or siderant. Again, a differential diagnosis can generally be made without any very great difficulty in fulminant cases, unless the symptoms happen to have an intermittent character, or occur in paroxysms, with intervals of tolerable comfort, as they did in Cases LVII. and LVIII. In such instances the physician who practises in malarial districts is always liable to suspect at first that the paroxysms of pain or restlessness may be due to malarial intoxication until the progress of the case reveals the true nature of the disease. The symptoms which attend epidemic cerebro-spinal meningitis are so different from those which belong to any other disorder, that not unfrequently the practitioner is freed from all embarrassment in diagnosing irregular or exceptional cases of it by the mere suggestion of its name. In other obscure cases he may at once get set right by recollecting that it is then prevailing as an epidemic. And, generally speaking, if the physician adopts the rule of taking all the symptoms into account, of subjecting each of them to a rigid interpretation, and of adopting only such an hypothesis to explain their occurrence as shall satisfactorily account for the presence of each and every one of them, he will never be much disconcerted in diagnosticating this disease. Finally, neither typhus nor typhoid fever should ever be mistaken for epidemic cerebro-spinal meningitis, because the former has no likeness to it in any respect except that each of them is produced by a specific poison, and the latter (typhoid fever) is always attended with characteristic phenomena which it is unnecessary to describe in this place.

Prognosis.—This disease is always very dangerous to life. The death-rate, however, varies greatly in different epidemics, and in different portions of the same epidemic. For example, in the Baden outbreak of 1864–5, out of 126 cases 38 died, or 30 per cent; in the Vistula epidemic of the same years Meissner estimates that there were in all 2000 cases, and more than 1000 deaths, or a mortality of over 50 per cent. (Vide *New Sydenham Soc. Retrospect*, 1865–6, pp. 56, 61.) According to Hirsch, the mortality ranged, in the various outbreaks which occurred between 1838 and 1865, all the way from 75 per cent on the one hand to 20 per cent on the other. The death-rate from this disease at the Hardwicke Hospital, in Dublin, in 1866, was not less than 80 per cent. At the Philadelphia Hospital, in the winter of 1866–7, it was only about 30; for Githens states that there were 161 cases, 43 deaths, 87 recoveries, and 43 still under treatment with a fair prospect of recovery when the report was made. In the Massachusetts epidemic of 1866 there were 278 cases and 170 deaths, which gives a mortality of 61 per cent. But the death-rate also varies considerably in different portions of the same outbreak. The first blows which are struck by this disease are generally the hardest. The first patients who are attacked by it are generally the sickest, and it is among them that the fulminant cases are usually found. In the irruption at Stanton Hospital our first case was destroyed in 5½ hours, our second in 24 hours, and our third in something over 48 hours. Rummel also observes that the disorder runs its course more quickly at the commencement than at the close of an epidemic. Hence, we say that the first cases of this disease are usually the severest, and that the death-rate is generally much higher in the beginning than in any other part of an epidemic.[1]

The ratio of mortality is greater in those under 14 years of age than in those between 14 and 35. After the last-named year the danger is again much increased. When life is prolonged beyond the fourth day, and fatal symptoms have not yet appeared, the gravity of the prognosis lessens considerably, for one half of all the deaths have happened before the fifth day, and one third before the third. Still, it must not be forgotten that in many instances death has occurred between the fifth and eighth days, and that some patients, after living five or six weeks, have then died just as recovery seemed probable. The prognostics which are the most unfavorable are a preternaturally slow pulse; rigid retraction of the head; general convulsions; incontinence of urine; albuminuria; coldness of the skin, with a diffused purplish hue; indifference as to the issue; paralysis of the tongue and pharynx, or inability to swallow; dilatation and insensibility of the pupils; and especially deep coma. Recovery after violent delirium is not uncommon; but when the patient has become profoundly comatose, there is scarcely any hope. All complications greatly aggravate the danger; those which especially do so being pericarditis, pneumonia, pulmonary collapse from obstruction of one or more of the bronchia, the formation of large bullæ with gangrenous spots, obstinate vomiting, and profuse diarrhœa. (*Tanner.*) Dr. Githens found a rapid fall in the temperature, as indicated by the thermometer, to be a sure precursor of collapse. Ziemssen noticed that very high temperatures (above 105°) were scarcely ever seen except in cases that terminated fatally. The favorable prognostics are, an abatement of the symptoms referable to the cerebro-spinal axis; general reaction, shown by the state of the heart, pulse, and skin; and a more natural respiration. There are many instances on record in which pregnant women got well. But the patient is not safe even when convalescence has fairly set in, for there is always risk of relapse, which in some epidemics has been constantly mortal. (*Clymer.*) The occurrence of a relapse, however, does not always indicate that the case will prove fatal, for Sanderson found in the Vistula epidemic that a case with relapses was generally destined to be tedious, but not fatal. (Vide *New Sydenham Soc. Retrospect*, 1865–6, p. 58.) Dr. Githens observed that a regular and gradual descent in the temperature indicated the beginning of convalescence. (Vide *American Journal of the Medical Sciences* for July, 1867, p. 38.)

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[1] In the New-York epidemic of 1872, the ratio of mortality, according to the reports of the health authorities, was considerably more than one half. At the same time, *Prof. Clark* observed in hospital patients a mortality of only one in four, or possibly one in three. (Vide *The Medical Record*, June 15th, 1872, p. 246.) May not this discrepancy be accounted for, at least in part, by the fact that a very large proportion of those who perish by cerebro-spinal meningitis die in the first twenty-four hours, and before they can be taken to the hospital? This view is strengthened by *Dr. Russel's* statement that in this epidemic the duration of the disease in fatal cases has seldom been more than twenty-four hours. Death, in the cases described by Dr. Morris, took place within twelve hours. (*Ibidem*, p. 246.)

When the acute symptoms have subsided, there is still a certain amount of danger from intense exhaustion during the tedious and irregular convalescence which frequently follows. Neuralgia, headache, dyspepsia, palpitation of the heart, stiffness of the neck, etc., will often retard the patient's progress. Relapses, too, as mentioned above, are not uncommon. In favorable cases, a period varying from fourteen days to six months may elapse between the day of attack and that of complete recovery. (*Tanner.*)

Treatment.—We are unacquainted with the specific poison on which the production of epidemic cerebro-spinal meningitis depends, and, therefore, the causal indications, as they are sometimes termed, are unknown, and can not be fulfilled, in the present state of our knowledge. We have not yet learned how to protect large populations from its outbreak, nor how to place the individual man beyond the influence of its specific cause. This difficult problem still awaits solution. We have elsewhere shown, however, that epidemic cerebro-spinal meningitis does not belong to the idiopathic fevers, but to the local phlegmasiæ; that essentially it is not a febrile disease, but a tropical inflammation of peculiar character. This piece of information concerning the nature of this disease is of great practical importance, because it is calculated to exert a large influence upon the plan of treatment. For example, if we happen to consider the disease to be a specific fever, we shall be apt to adopt an expectant plan of treatment, and aim to guide rather than arrest its course; but if, on the other hand, we hold it to be a local inflammation, we will treat it on far different principles. Now, holding as we do, and with good reason too, as we trust we have shown in the forepart of this chapter, that epidemic cerebro-spinal meningitis is a local phlegmasia due to a specific cause, the latter, however, so far as we now know, being entirely beyond our control, the indications to be fulfilled by treatment appear to us to be but few and plain. They are *first*, to arrest or abort the inflammatory process in the cerebro-spinal meninges and in the contiguous parts; and *second*, to obviate or remove the consequences of this process which usually appear in the form of serous, fibrinous, or purulent exudations within or upon the cerebro-spinal meninges.

Again, with what remedial measures shall we strive to fulfil the first indication? Niemeyer has given a fit answer to this question. He says: "Just as in other malignant and fatal epidemic diseases, we must consider those cases only in which there is some slight hope of recovery, in deciding what remedial measures are the best. Whoever tries any plan of treatment that may be proposed on only the severest cases of epidemic cerebro-spinal meningitis will attain negative results by any procedure. The treatment customary for sporadic meningitis, consisting of the energetic use of cold in the form of ice-compresses or the ice-bag kept on the head, the application of leeches or wet cups behind the ears, and the internal administration of calomel, is also advisable in epidemic cerebro-spinal meningitis, as is very evident from its excellent effect in patients attacked with the premonitory symptoms of the disease, severe headache and pain in the neck, during an epidemic. But, according to most observers, this mode of treatment has preserved its reputation even in marked cases of the disease; there is but little opposition to it, and even this is based on its want of success in the severest cases. I have," says Niemeyer, "no personal experience with morphia in this disease, but a number of trustworthy observers, Ziemssen and Maunkopff among others, speak most favorably of it, especially when administered by subcutaneous injection. Ziemssen says: 'Although we have used morphia frequently, we have never seen any injurious effects from it, but, on the contrary, such a decidedly palliative action that, along with cold, it seems the most indispensable remedy in the treatment of [epidemic] meningitis.' Almost all observers agree that quinine is entirely useless, even in decidedly intermittent cases." (Vide *Text-Book of Practical Medicine*, vol. ii. p. 225, 1st Am. ed.) According to Niemeyer, then, the best remedies of an abortive character are the application of cold to the head and spine, by means of frozen compresses or the ice-bag, the abstraction of blood from behind the ears and the nape of neck by leeches or cups, the internal administration of calomel, and the hypodermic injection of morphia. With regard to the local abstraction of blood, etc., it seems to be Burdon-Sanderson's opinion, also, that in some cases which he saw the leeching, etc., did real and even striking good, though in many others it had no effect. (Vide *New Sydenham Soc. Retrospect*, 1865–6, pp. 61, 62.) Dr. Githens remarks that the application of a few wet cups to the temples and neck uniformly relieved the headache in the cases treated at the Philadelphia hospital in the winter of 1866–7. There is, then, good reason to believe that the abstraction of blood, in reasonable quantity, by leeches or cups, from the temples, or from behind the ears, or from the nape of the neck, according as the cerebral or the spinal evidences of local mischief happen to predominate, generally proves a safe, and not unfrequently a very useful remedy, especially in the early stages of this disease. With regard to the employment of ice-cold, Dr. Burdon-Sanderson recommends that it should be applied to the spine during the first day, and Dr. J. Netten Radcliffe says: "The application of cold to the head and spine, either by means of ice or a freezing mixture, in Esmarch's (or Chapman's) India-rubber bags, has furnished by far the most satisfactory results of all direct treatment." If there is much prostration during the local use of cold, the trunk and extremities should be kept warm by cotton-wool, hot sand-bags, or hot-water bottles. (*Clymer.*) We think there is no doubt that the energetic use of cold on the head or spine, or in both situations, as either one or both of these parts chance to be especially affected by the disease, is a most valuable means for subduing the inflammation. Again, opium or morphia has been greatly extolled, and the late Dr. Valleix says that, in large doses, it is the only drug which has appeared to exert any real influence over the disease; and this good opinion of its effects is confirmed by Minor, Tully, Hirsch, Boudin, Forget, Levy, Boutin, C. Broussais, W. H. Draper, Levick, Stillé, and others. (*Clymer.*) Opium or morphia, when freely administered, appears to exert the same restraining or curative influence on inflammation of the cerebro-spinal arachnoid, that it does on inflammation of the other serous membranes, for instance, peritonitis, pleuritis, and pericarditis. A grain of opium, or gr. $\frac{1}{6}$ of morphia, should be given every hour in severe cases, and every second hour in less severe ones. In many cases it would be better to administer morphia subcutaneously, as recommended by Niemeyer. We believe that the value of opium or morphia, as a remedial measure in the early stages of this disease, has not been over-estimated. The utility of calomel, however, is not so clear as that of opium. The German physicians employed it in large doses during the late epidemic, and some of them at least appear to be satisfied with its effects. When judiciously prescribed for young and vigorous subjects, it probably does more good than harm in epidemic cerebro-spinal meningitis. With regard to quinia, the experience of many observers shows that it exerts no influence over the inflammatory process in the cerebro-spinal meninges, and that the fever seldom rises so high in this disease that it can do any good as a febrifuge. But, in cases where there is a malarial complication, and generally in those which occur in paludal districts, its exhibition should be recommended. A few doses of five or ten grains each, given at the outset, will generally suffice to free the case from its malarial features. Quinia, however, is not a specific for this disease; for one of our patients was attacked with it while taking five grains of this remedy four times a day. (See Case LVIII.) Finally, counter-irritation can, not unfrequently, be employed with advantage. After blood has been drawn by leeches or cups, a blister should generally be applied to the nape of the neck. There is also much strong testimony in behalf of counter-irritation and blistering along the spine. The actual cautery freely applied to the back, has been followed by great alleviation of the pain and other symptoms. (*Rollet.*) At the outset of an attack, stimulating embrocations to the spine and extremities, and moist or dry heat to the limbs, have been much employed and praised by physicians of all countries, and are without doubt often very relieving. (*Clymer.*) Various stimulating and anti-spasmodic medicines, such, for example, as brandy, ammonia, camphor, valerian, and musk, have been administered for the purpose of palliating the symptoms of cerebro-spinal depression and irritation which result from the injury done to the great nerve-centres by the inflammatory process and its products; they but seldom or never do any good and may do much harm, and, therefore, should generally not be used in this disease. The sulphite and bisulphite of soda have also been administered with a view to produce an antizymotic or catalytic effect upon the blood, but without benefiting the patients, and no reliance should be placed upon them. In fine, the only remedies which have appeared to do any good in cases of this disease have been such as allayed the inflammatory process in the cerebro-spinal meninges, etc., on the one hand, or assisted in getting rid of its products, on the other. Those which constitute the first-named class are not very numerous; they are local blood-letting, the energetic application of cold to the head and spine, the employment of counter-irritants, and the liberal use of opium or morphia, combined in some instances with quinine, in others with calomel, but in most cases given by itself. Those which constitute the other class are still less numerous, and chief among them is the iodide of potassium. In cases where a copious exudation of serum has occurred, and produces dilatation of the pupils and other symptoms of general paralysis, this medicine may sometimes be administered with great advantage for the purpose of procuring absorption, just as it has been given in analogous cases of hydrocephalus. When it is administered with this end in view, the doses should be large, and the intervals between them short. To a child of six or eight years, five grains should be given every three or four hours; to an adult, double that quantity. The tincture of iodine also may sometimes be applied as a paint over the spine and on the shaven scalp, with benefit, in such cases.

The complications and sequelæ of epidemic cerebro-spinal meningitis must be treated on the usual principles. Quinine, belladonna, bromide of potassium, iodide of potassium, and iron appear to be the remedies which have been most frequently required.

When the patient survives the first onset of the disease, his alimentation, or supply with nourishment, not unfrequently becomes an important consideration or point in the treatment. While he lays delirious and more or less completely unconscious, his physician should see that nutritious and suitable articles of food, such as beef-tea, milk, eggs, farina, or oatmeal-gruel, are given to him at short intervals and in sufficient quantity, not only to prevent his dying of starvation, but also to sustain his strength while the reparative forces of his system shall overcome the consequences of the meningeal inflammation.

The experience of the New-York outbreak of 1872 has confirmed the value of nearly all of the remedial measures mentioned above, but especially the value of topical bleeding by leeches or cups, the application of cold to the head, etc., and the early administration of opium or morphia in full doses, by the stomach or hypodermically. Bromide of potassium also proved of decided service, but not in connection with opium, in quieting restlessness. When the body-heat rose to 103° or more, the application of the wet-pack was decidedly useful in abating the fever. *Professor Clark* found that the temperature could be lowered in this way to 101° Fahr. (Vide *The Medical Record*, June 15th, 1872, p. 246.) After the first stage, rapid blistering of the nape of the neck and posterior spinal region with cantharidal collodion generally proved beneficial.

Finally, when this disorder appears, either as an epidemic or as a local outbreak, all the sanitary measures, commonly used in other affections of a common origin, should be put in force. Mr. J. Simon, however, says: "I am strongly of opinion that the best sanitary precaution which, in the present state of our knowledge, can be taken against the disease, must consist in care for the ventilation of dwellings."

The history of the New-York epidemic of 1872, however, shows that the following preventive measures should be taken: 1. The house-drainage should be rendered so complete that foul sewer-gases can not be diffused in dwellings. 2. The land-drainage, especially that of made ground, should be improved as much as possible, and damp tenements should not be occupied. 3. Over-crowding, especially in tenement-houses, should not be allowed; and the garbage, ordure, and other refuse matter, so apt to accumulate in these localities, should be promptly removed. 4. House-ventilation should be made as nearly perfect as possible. By fulfilling these four plain indications, much may be done to prevent the development and arrest the spread of epidemic cerebro-spinal meningitis.

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